THE POLITICAL ECONOMY OF LIFE AND DEATH IN THE U.S.A.

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December 2018

This dissertation is submitted for the degree of Doctor of Philosophy.

Declaration

This dissertation is the result of my own work and includes nothing that is the outcome of work done in collaboration, except as declared in the Introduction. I further state that my dissertation is not substantially the same as any that I have submitted, or, is being concurrently submitted for a degree or diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Introduction and specified in the text. I further state that no substantial part of my dissertation has already been submitted, or, is being concurrently submitted for any such degree, diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Introduction and specified in the text. The dissertation does not exceed the prescribed word limit for the relevant Degree Committee.

Abstract

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This research charts the structural transformations of American capitalism and attendant shifts in the distribution of health and illness at the dawn of the 21st century. Through the analytic lens of the political economy of public health, I shed novel light on the upstream determinants of America's overdose epidemic, which is claiming tens of thousands of lives every single year, and on deepening social inequalities in life expectancy, all-cause mortality, and premature mortality risk. Economic decline, notably in the form of deindustrialisation, has fractured working class communities and spawned a highly stratified social structure. In response to this development, American social policy has undergone a distinctive transformation involving a historically unprecedented expansion of the penal system. This has resulted in the rapid swelling of the correctional population, reaching almost 7 million people in 2012, of whom over 2.2 million find themselves behind bars. Despite the salience and magnitude of these phenomena, virtually no existing research has investigated the connections between deindustrialisation, incarceration,

and America's public health crisis. This thesis fills this gap by using new data from U.S. states and counties between 1980 and 2014 to examine the social, economic, and political roots of increasing health inequality.

Acknowledgments

I dedicate this thesis to my parents, Shohreh and Fariborz, for their unwavering love and support, for their patience and confidence.

Thank you, first and foremost, to my supervisor, Professor Lawrence King, for his generosity, kindness, and unmatched mentorship. It would be foolish to pretend that I would have been able to envisage and operationalise – not to mention complete – the present research without his guidance and expertise. I am deeply grateful for everything he has taught me, for the time he has devoted to me, and for the professional connections he has made possible. I look forward to continuing our work together in the years to come.

My special gratitude goes to Michael Marmot, for his generous support and collaboration, and to my other co-authors, Michael Ash, Jacob Kang-Brown, and Martin McKee, who have helped me improve this research. Their indispensable contributions are gratefully acknowledged and appreciated.

Thank you to Ann Louise Kinmonth, Mike Kelly, and the rest of the St. John's Reading Group on Health Inequalities, for offering a unique site of

interdisciplinary study and debate, for inviting me to present my work to the Group, and for their kind support.

Thank you to Sarah Hawkes, for her persistent kindness and encouragement. Thank you to Adel Daoud for many a fruitful discussion of quantitative methodology, and for his friendship. And thank you also to Thomas Forster, for the mathematical lunches and puzzles.

Thank you to Mona and Cele for their support and companionship, and for our truly memorable year together in Cambridge. And thank you to Laura – for being who she is.

Last but most certainly not least, I thank my two uncles from far away, Mohammad and Farhad, who know the meaning of imprisonment but who, to me, embody nothing but freedom.

Contents

Introduction

In 2017, over 72,000 individuals, largely from the bottom of the class structure, died from drug overdoses in the United States (National Institute on Drug Abuse, 2018), joining the ranks of over half a million people who have suffered the same fate since 1980 (Dwyer-Lindgren et al., 2018). Since the turn of the previous century, the richest Americans have experienced gains in longevity equivalent to the curing of cancer, whilst the health of the poor has either stagnated or declined (Olshansky et al., 2012; Chetty et al., 2016). These striking facts form the starting point of this doctoral thesis, which seeks to offer a novel social scientific account of their root causes. The purpose of this introduction is to situate my research agenda in an emerging body of literature that tries to grapple with the reality of the American population health landscape. In what follows, I summarise the state of current knowledge, identify important gaps, and outline the analytic steps through which I seek to make a contribution to this field of enquiry.

Explaining America's public health crisis

In a recent pair of studies, subject to extensive media coverage and public debate, Anne Case and Angus Deaton examine mortality trends in the United States since the tail end of the previous century (Case and Deaton, 2015; 2017). Their analyses yield four principal findings. First, they note a dramatic rise in mortality rates from so-called "external" causes such as drug-related poisonings, intentional self-harm, accidents, and alcohol-induced liver cirrhosis. This rise has been particularly salient amongst non-Hispanic Whites aged between 45 and 54, who have experienced an average annual increase of 5.4% in such deaths between 1999 and 2015. Second, they highlight how so-called "deaths of despair" constitute an increasingly central component of all-cause mortality, resulting in a sudden reversal of overall mortality trends for non-Hispanic Whites in midlife. They estimate that this reversal has caused 488,500 excess deaths between 1999 and 2013, and 54,000 excess deaths in 2013 alone. This lethal pattern has continued its upward-spiralling trajectory after 2013 and is further associated with stagnating survival rates from heart disease and cancer. Third, whilst these patterns exist for the population as a whole, the educational health gradient has increased sharply in a short time period. For instance, amongst non-Hispanic Whites, the ratio of mortality rates of those with no more than a high school diploma to those with a university degree has shifted from 2.6 in 1999 to 4.1 in 2013 for all causes, from 2.4 to 4.0 for all external causes, and from 4.0 to 7.2 for drug-related poisonings alone. Those at the bottom of the educational hierarchy have experienced an increase of 134 additional all-cause deaths per 100,000 population since 1999, of which 68.7 have been due to external causes, of

which, in turn, 44.3 have been overdose deaths. Finally, Case and Deaton show that increases in midlife mortality are accompanied by rising morbidity, notably when it comes to self-reported experiences of chronic pain and psychological distress.

These findings offer a series of scientific puzzles: what are the root causes of rising "deaths of despair"? What are the driving forces behind increasing health inequality between the top and bottom of the socioeconomic order? And why have these patterns, especially when it comes to overdose deaths, materialised at such a large scale in the United States but not in other wealthy countries? The purpose of the present research is to address these points through empirical analysis and theoretical articulation as seen through an analytic lens located at the interface of sociology, political economy, and public health. Whilst the studies described above constitute an important starting point, I will seek to deploy them as an empirical springboard to probe the deeper determinants of inequalities in disability, disease, and death in 21st century America. Case and Deaton (2017) do offer a preliminary explanatory framework in which income stagnation, downward intergenerational mobility, and fractured social and family relations form the key ingredients of what they dub "cumulative disadvantage". In particular, they highlight long-run patterns of deteriorating employment parameters, notably declining labour force participation rates amongst individuals with low levels of formal education, as well as unstable social bonds resulting from economic hardship, especially in the form of dwindling marriage rates and disintegrating family units.

In what follows, I draw on the insights of Case and Deaton but offer

an alternative aetiological account of America's ongoing public health crisis. In particular, my divergence rests on three simple observations. First, whereas Case and Deaton focus exclusively on a specific subset of the American population as defined by ethnicity and age (i.e. non-Hispanic Whites between the ages of 45 and 55) within a timespan of about 15 years, emerging evidence suggests a broader pattern of declining health at the bottom of the class structure that is neither confined to this specific demographic nor to the given time period. In fact, nation-wide age-standardised mortality rates from drug use disorders (to pick the most dramatic example) increased by 618.3% between 1980 and 2014 as a whole – amounting to no less than 542,501 deaths – but by 238.2% *before* the new millennium and by 112.4% from 2000 to 2014 (Dwyer-Lindgren et al., 2018). Growth in the latter period has been linked to increased opioid abuse since the late 1990s. In December 1995, the U.S. Food and Drug Administration approved the deregulation of OxyContin,[1] a medication used for the relief of moderate to severe pain. Research has shown that unequal clinical prescription patterns by patient ethnicity have disproportionately "favoured" non-Hispanic Whites (Pletcher et al., 2008; Anderson et al., 2009; Singhal et al., 2016; Netherland and Hansen, 2017). However, concurrent increases in drug-related mortality for African Americans have been driven by heroin on the one hand – currently rising at an annual rate of 34% – and synthetic opioids, such as fentanyl and its analogues – rising at an annual rate of 107% – on the other (Alexander et al., 2018). A recent report by the Chicago Urban League revealed

[1] Produced by Purdue Pharmaceutical, whose sales revenues from OxyContin alone amounted to $35 billion by 2017 (Keefe, 2017).

that the rate of opioid overdose deaths amongst African Americans is higher than that of the general population in several states, including District of Columbia, Illinois, Minnesota, Missouri, West Virginia, and Wisconsin. In Illinois, deaths from pain pills increased nearly nine-fold amongst African Americans and tripled amongst Whites. In the city of Chicago – one that has experienced wrenching deindustrialisation and economic decline at the bottom of the sociospatial structure – nearly half (48.4%) of all opioid deaths occur amongst African Americans, despite the fact that Blacks make up only 32% of the total population. The majority of these deaths take place in Chicago's stigmatised neighbourhoods of concentrated poverty of the former industrial Black Belt, including Austin, East and West Garfield Park, Englewood, Fuller Park, Humboldt Park, and North Lawndale (Bechteler and Kane-Willis, 2017). As such, the exclusive focus on the existential crisis of America's middle-aged Whites seems to shift attention away from a more deep-seated tendency of class-driven "despair" that cuts across ethno-racial lines of demarcation (see also Woolf et al., 2018).

Second, mounting overdose deaths remain subject to substantial spatial variation, as visualised in Figure 1, which maps the distribution of age-standardised mortality rates from drug use disorders at the U.S. county level (Dwyer-Lindgren et al., 2018). The reader will note how both levels and changes in such deaths follow a distinct geographical pattern, notably in the form of prominent clusters in Kentucky and West Virginia, in New Mexico, but also in the former slave states of the South. However, such variation is either suppressed or reconfigured when bundling together various causes of death under the single rubric of "despair" (see Case and Deaton, 2017:

409–410). As more recent data released by the Institute for Health Metrics and Evaluation reveal, the geography of the overdose epidemic differs significantly from that of alcohol- or suicide-related deaths (Dwyer-Lindgren et al., 2018). The source of this spatial expression is far from evident in the extant literature[2] and rigorous empirical studies of geographical heterogeneity remain scarce. Whilst economic disadvantage is likely to play a major role at the aggregate level, there is little evidence of why such disadvantage proves lethal in some places but not others. This puzzle is reinforced in a recent study by Raj Chetty and colleagues (2016), who examined the relationship between income and life expectancy at age 40 in the United States between 2001 and 2014. They demonstrate that the life expectancy gap between the top and the bottom of the income spectrum has increased rapidly at the dawn of the century, to the point where indigent lives are cut short by up to a decade and a half compared to those of the wealthy. However, whereas the rich tend to live longer everywhere, life expectancy amongst the poor differs markedly from one place to another, as seen in Figure 2. To the areas of poor health highlighted in the previous maps, this one adds the Midwestern Rust Belt, where rapid industrial decline has left an indelible social imprint. In other words, for those tethered to the bottom rungs of the socioeconomic order, geography matters. When viewed in tandem with Figures 3 and 4, which visualise the distribution of levels and changes in life expectancy across counties based on novel data from the Institute for Health

[2] It is worth highlighting that, contrary to many media portrayals, this spatial expression does *not* correspond to a divide between urban and rural areas. This is also emphasised by Case and Deaton in their latest Brookings paper.

Metrics and Evaluation, Chetty et al.'s analyses offer strong evidence of a correspondence between social and physical space. This correspondence merits scientific probing – with a focus on the specific *components* of disadvantage that may prove deadly, and how they relate to one another.

Finally, although the widespread emphasis on economic decline and insecurity offers a largely convincing causal narrative surrounding the ongoing opioid epidemic, it fails to explain why resource shocks have proven so fatal on one side of the Atlantic but not on the other. As evidenced by Figures 5 and 6, the skyrocketing of preventable mortality in America is unparalleled elsewhere in the world – although fiscal austerity in post-recession Europe has done more than expected to offer a sense of balance (Stuckler and Basu, 2013; Kentikelenis et al., 2014; Reeves et al., 2015; Hiam et al., 2017). What is different about the United States? Towards the end of their Brookings paper, Case and Deaton outline a simple model of general health as a function of some latent variable.[3] Their provisional application of this model to their data on mortality trends over time leads them to conclude that at the root of America's public health crisis, "there may be *two* underlying factors, not one, but they are not very different" (Case and Deaton, 2017: 438; emphasis added). What second factor other than economic decline could account for the phenomena described above?

One of the largest and most astounding changes taking place in the United States over the past four odd decades has been the growth of the penal state

[3] Their model takes the form $y_{ab} = \alpha + f(a) + \theta X^b$, where y is the health outcome variable; a and b index age and birth year, respectively; f is some unknown functional form; α, X, and θ designate an intercept term, some unobservable initial condition exposure, and its corresponding systematic component parameter, respectively.

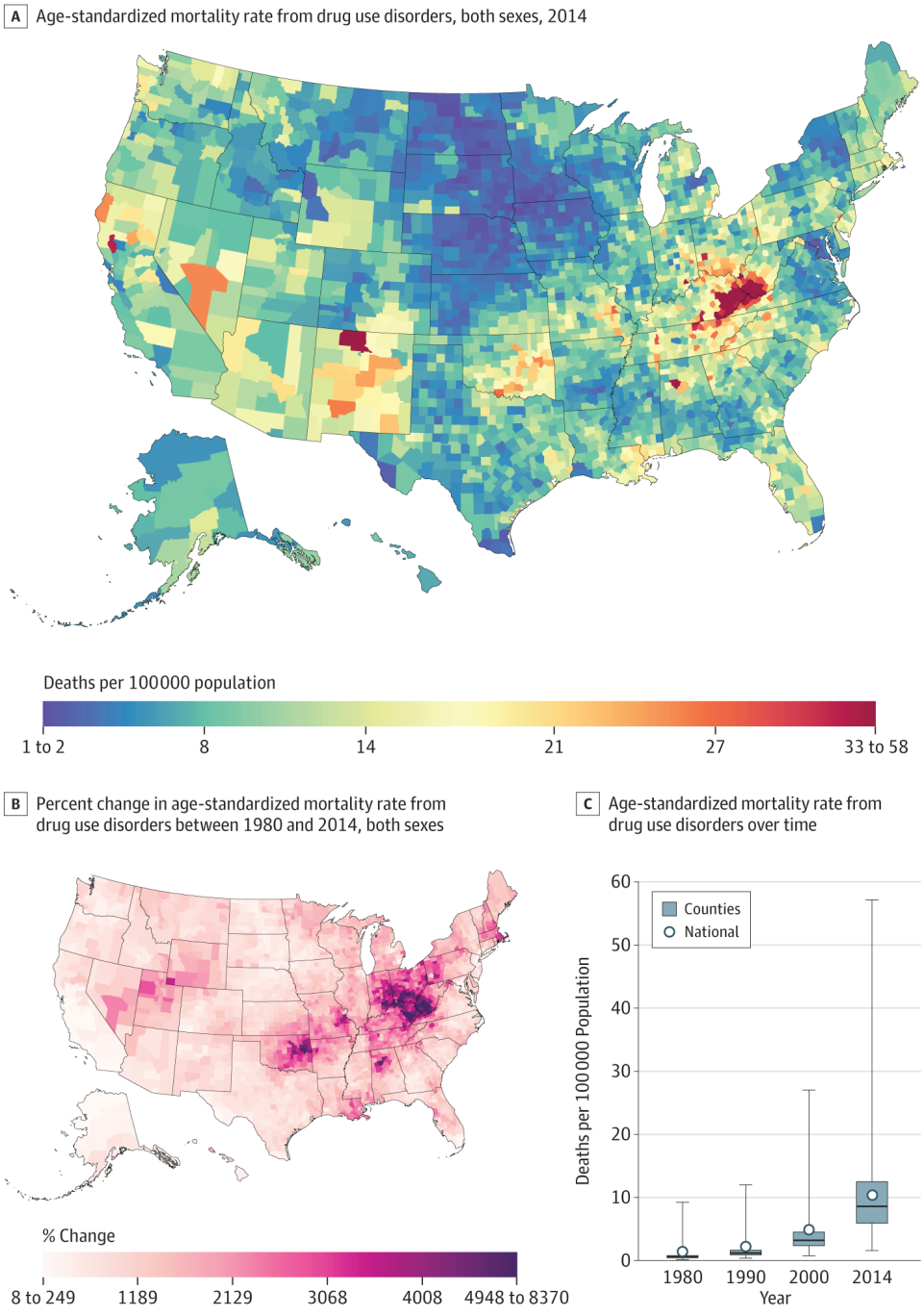

Figure 1: *Spatial variation in age-standardised mortality rates from drug use disorders at the U.S. county level. Source: Dwyer-Lindgren et al. (2018).*

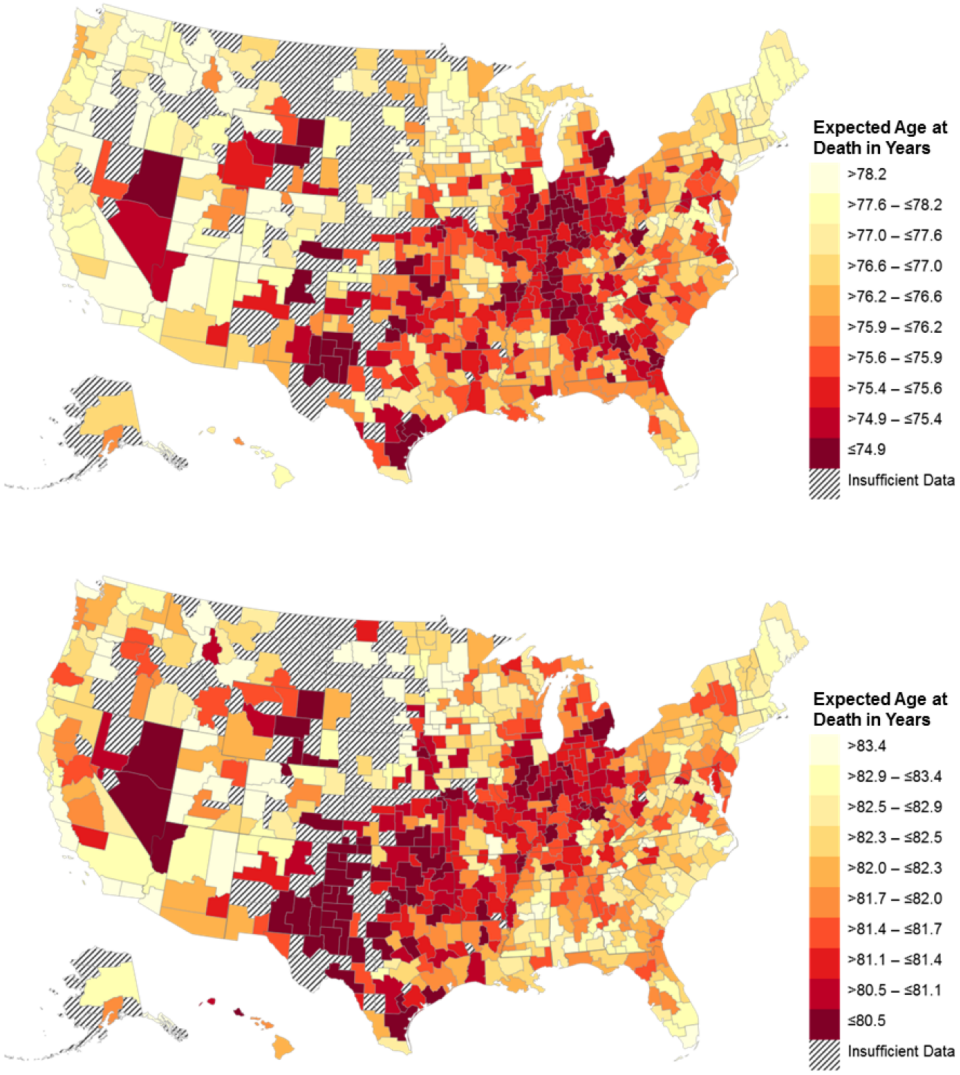

Figure 2: *Spatial variation in male (top figure) and female (bottom figure) life expectancy at age 40 in the bottom income quartile at the U.S. commuting zone level. Source: Chetty et al. (2016).*

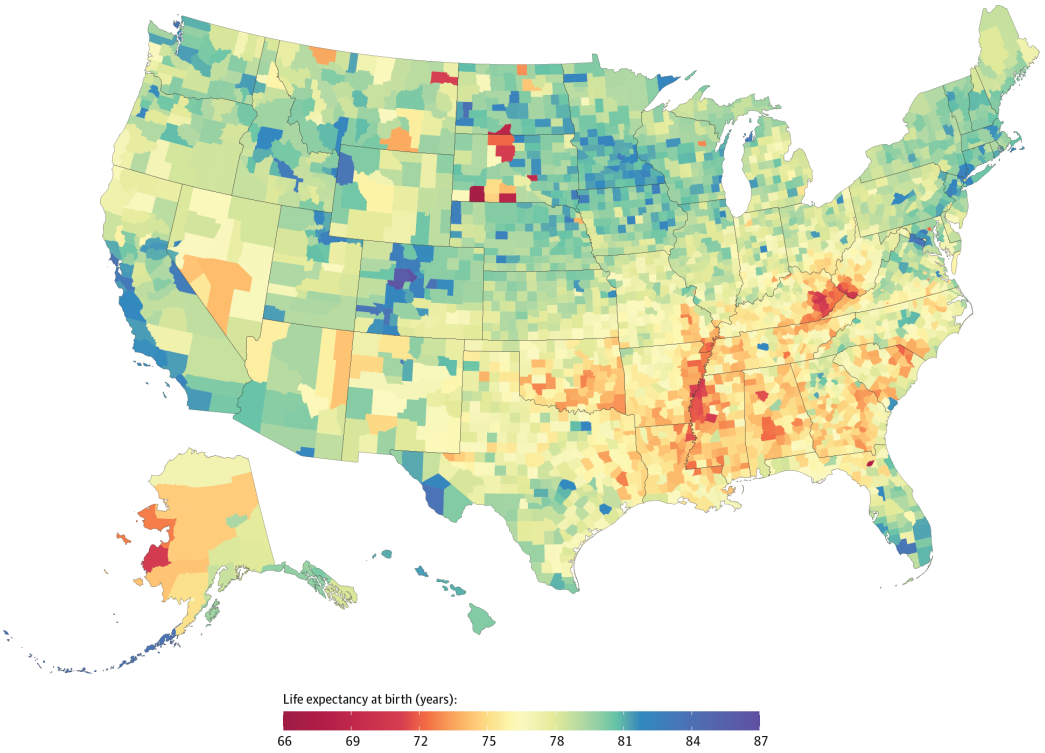

Figure 3: *Spatial variation in life expectancy at birth at the U.S. county level. Source: Dwyer-Lindgren et al. (2017).*

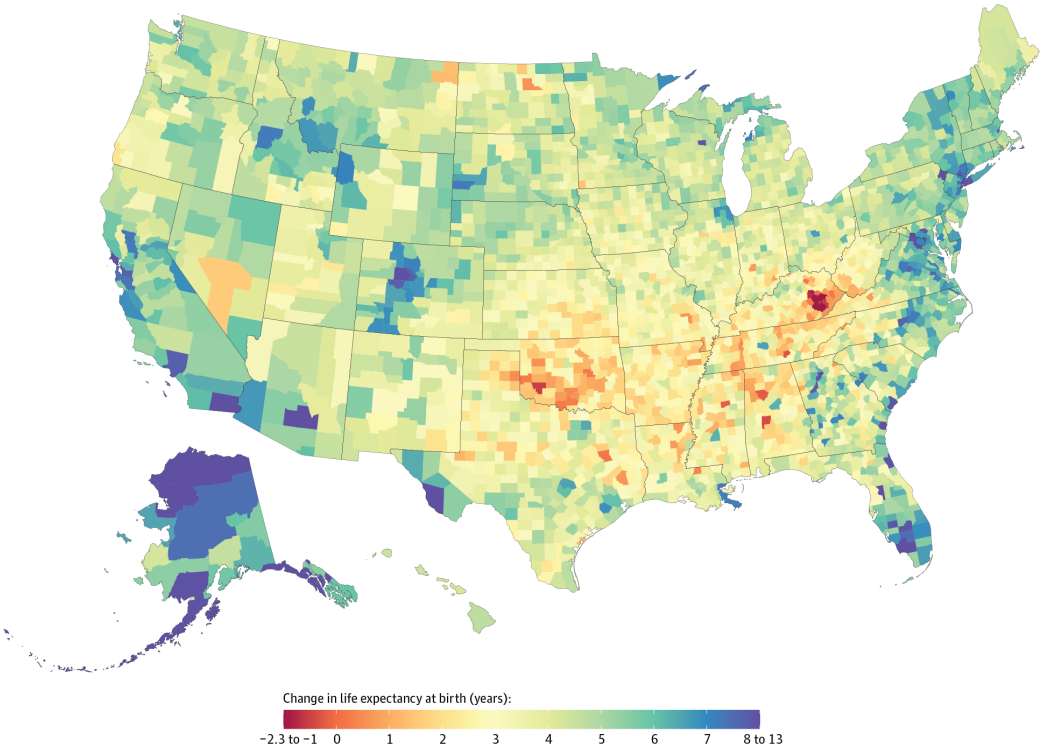

Figure 4: *Spatial variation in changes in life expectancy at birth at the U.S. county level between 1980 and 2014. Source: Dwyer-Lindgren et al. (2017).*

(National Research Council, 2014). After 50 years of relative stability, the national jail and prison incarceration rate stood at 161 residents per 100,000 population in 1973. In 2007, the corresponding figure was 767. In absolute numbers rather than rates, this amounts to a shift from just under 400,000 to over 2.2 million individuals behind bars – a sevenfold increase in less than four decades. After a slump in the 1960s, state level growth in prison incarceration rates exceeded 60% in the 1970s, 100% in the 1980s, 55% in the 1990s, and around 10% in the 2000s. This development alone is not only historically unprecedented but has also left the United States in a unique position on an international scale, as illustrated in Figure 7, which compares prison incarceration rates amongst selected countries.[4] However, as visualised in Figure 8, the incarcerated population accounts for only a quarter of those under criminal justice supervision when individuals subject to probation and parole are included (Wacquant, 2009: 63). In the last four decades, the probation population has grown fourfold from around 920,000 to over 4 million, whilst the population under parole has grown by a factor of six from 143,000 to 851,000 in 2012. Thus, the overall correctional population has experienced rapid swelling since 1972, counting just under 7 million individuals in 2012 (National Research Council, 2014: 40–42). Approximately 3% of the total adult population and 15% of adult African American males have ever been imprisoned, and around 8% of all adults and a full one-third of African Americans have felony records, amounting to almost 19 million

[4] A simple comparison of Figures 5 and 7 offers a first clue of a central argument in this thesis. The reader will note how the distribution of drug-related mortality seems closely linked to the size of the penal state.

people nationwide (Shannon et al., 2017).

What these numbers conceal is the twofold selectivity whereby the penal state operates across both social and physical space. Amongst those condemned to serve time in American prisons, unemployment rates lie just below 40%, whilst 15% work part-time or occasionally; around 13% possess any post-secondary education or vocational training, whilst half the population failed to graduate from high school; two-thirds belong to households living on less than half the official poverty line for a family of three, yet less than 25% received any form of public assistance; up to 60% were raised by a single parent, whilst 14% lived in an orphanage or group home as a child (Wacquant, 2009: 70–71). The class disproportionality of exposure to penal confinement is evidenced by how America's punitive upsurge has more or less exclusively been concentrated amongst those without any form of higher education. In fact, whilst ethno-racial disparities in imprisonment have been at the forefront of both media and scholarly attention (see e.g. Alexander, 2010; Pettit, 2012), changes in incarceration over time suggest even more pronounced class differentials (see National Research Council, 2014: 65–67; see Pettit and Western, 2004; Western, 2006; Wacquant, 2010; Chetty et al., 2018: Figure VII; for a discussion of "racial fluidity" across the class structure, see Saperstein and Penner, 2012). Between 1972 and 2010, the ratio of incarcerated men aged 20–39 with no more than 12 years of completed education to those with at least some college attendance jumped from around 2.0 to over 8.0 amongst both Blacks and Whites.[5] On the other hand, the Black-

[5] The social structure of the incarceration gap is even more astounding when examining the trajectories of high school dropouts. Amongst African Americans, the ratio of incarcerated men with less than 12 years of completed education to those with at least some

White imprisonment ratio amongst those with no more than a high school degree has remained relatively stable at around 4.0. When comparing birth cohorts of individuals born in 1945–1949 on the one hand and those born in 1975–1979 on the other, estimated probabilities of imprisonment over the life course (as opposed to at single time points) reveal that for those with no post-secondary education, the cumulative incarceration risk has grown about twofold – from 10.2% to 18.0% – for Blacks and about sixfold – from 0.7% to 4.1% – for Whites (Western and Wildeman, 2009: 231). Moreover, in recent years, the racial gap in imprisonment has narrowed somewhat due to declining incarceration rates amongst Blacks – whose middle- and upper-class fractions have become increasingly differentiated, thus escaping the clutch of the penal apparatus (Wacquant, 2010) – and rising incarceration rates amongst (poor) Whites, who have experienced steady growth in confinement in the past couple of decades. This is especially true of the White jail incarceration rate, which has nearly doubled from 135 to 255 inmates per 100,000 population between 1990 and 2013 (Subramanian et al., 2018).

Spatial variation in carceral inflation ranges from twofold increases in states such as Maine or Massachusetts to sixfold growth in states such as Louisiana and Mississippi (Western, 2006: 42). Before the turn of the cen-

college attendance has galloped from around 3.0 to over 17.0 between 1972 and 2010. For Whites, the corresponding figures are roughly 2.0 and 25.0 (see National Research Council, 2014: 65). It is worth noting, however, that the number of high school dropouts as a share of the total population has undergone significant changes in recent decades, whilst the share of those with no more than a high school degree has remained more or less stable at approximately 40%, which is why data on the latter group are presented in the main body of the text.

tury, between 1972 and 2000, the former two states experienced imprisonment growth equivalent to between 150 and 200 additional prisoners per 100,000 population, whilst the latter two states added over 600 prisoners per 100,000 (National Research Council, 2014: 43). By the end of 2015, states like Maine, Massachussetts, Minnesota, Rhode Island, and Vermont held around 300 individuals in local jails or state prisons per 100,000 population, whereas states like Alabama, Louisiana, Mississippi, and Oklahoma held roughly 900 individuals per 100,000 population (Keable and Glaze, 2016). Nested within states, urban areas of concentrated disadvantage, such as Chicago's aforementioned territories of urban relegation, experience incarceration rates more than forty times higher than privileged White communities and three times higher than communities with a similar crime rate (Sampson and Loeffler, 2010: 27–28; see also Sampson, 2012). Similarly, in the wake of economic desolation, Detroit's deindustrialised wasteland has undergone a social transformation so profound that the number of its inhabitants under correctional supervision outweighs the number holding union jobs in the city's manufacturing plants (Thompson, 2010: 708).

The above snapshot of the sociospatial dynamics of the American carceral state strongly suggests the need to study economic decline and penal expansion – and their potential role in the overdose epidemic – in tandem rather than in isolation from one another. A rich body of evidence (to be reviewed in the coming chapters) ties incarceration to a variety of factors that are associated with drug overdoses and, more broadly, with inequalities in health (Link and Phelan, 1995), such as stigma, joblessness, family disruption, and neighbourhood decline (for a recent study, see Western [2018]). Nonetheless,

virtually no existing research has sought to link the over half a million drug-related deaths that have occurred over the past three and a half decades or the rapid increase in the health gap between the top and the bottom of the social order to the gargantuan expansion of the penal state since the early 1970s. The penal state, despite its unique place in American society, has remained conspicuously absent from research and policy debates surrounding "deaths of despair" and health inequality. Indeed, in the 80-page Brookings document containing both Case and Deaton's latest analysis and supplementary comments and debates, including lengthy responses by two prominent economists, David Cutler and Adriana Lleras-Muney, *not a single mention* is made of one of the most singular and consequential developments in American social policy in recent decades as a potential cause of declining health and deepening health inequality.

My argument, which I shall theoretically expound and empirically substantiate in the following chapters, is twofold. On the one hand, I leverage insights from previous research to investigate the role of resource shocks – such as job destruction and income decline – in driving the rise of America's public health crisis. On the other, I argue that such resource shocks must be viewed in tandem with the set of institutions that abet or abate broader structures of inequality. More specifically, I propose that the distinctive feature of the American case is a shift in political emphasis from the protective to the corrective wing of the policy apparatus, of which the most pronounced manifestation is the historically unprecedented expansion of the penal state. Research has established that the rise in incarceration was not merely the result of increases in crime rates (Wacquant, 2009; National

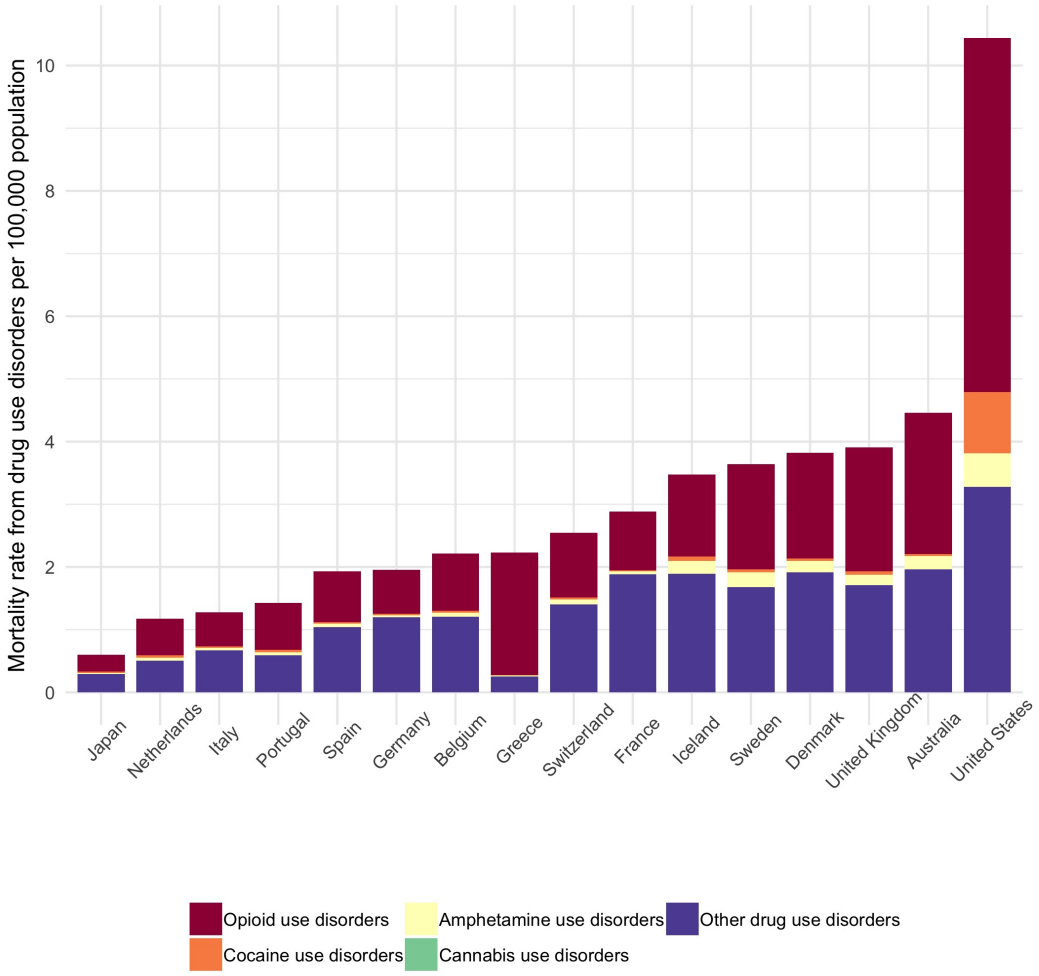

Figure 5: *Cross-national differences in mortality rates from drug use disorders between selected countries in 2016. Data source: Institute for Health Metrics and Evaluation.*

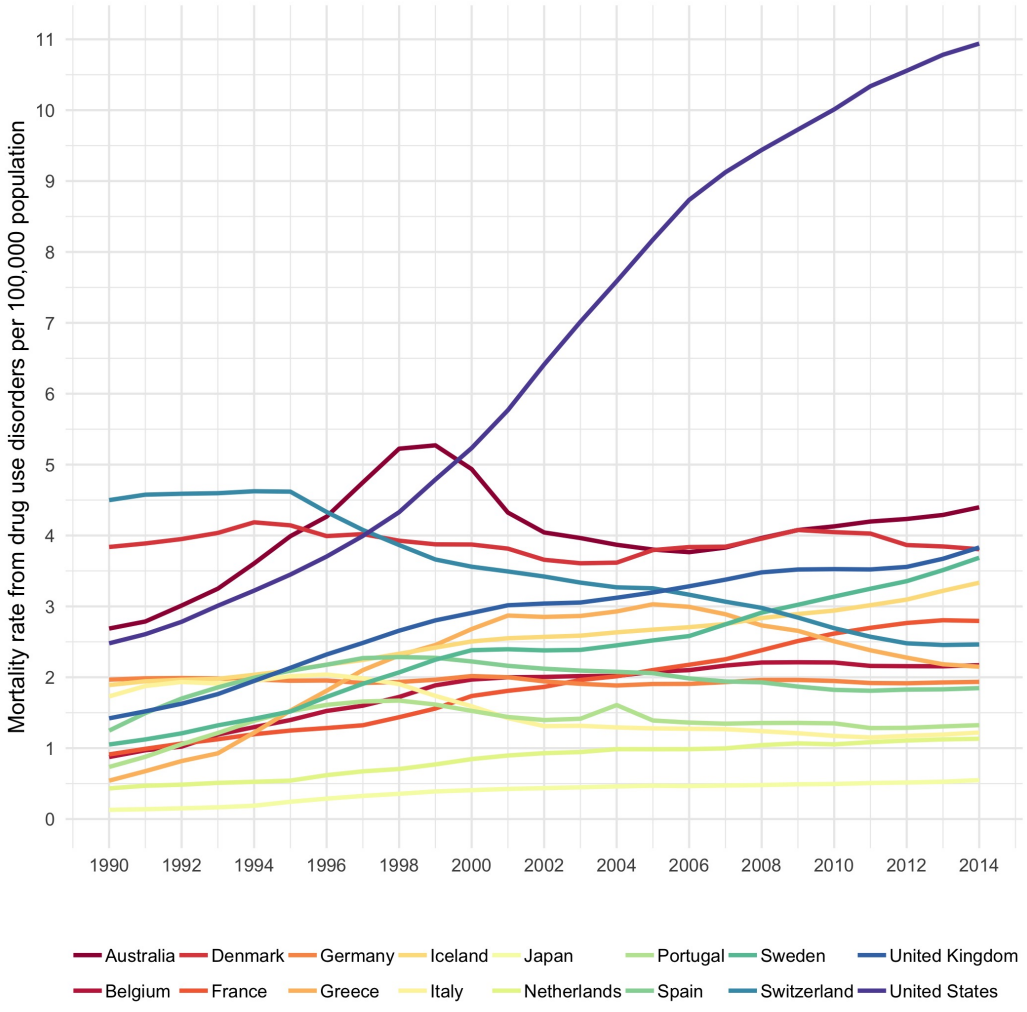

Figure 6: *Cross-national differences in drug-related mortality trends between selected countries between 1990 and 2014. Data source: Institute for Health Metrics and Evaluation.*

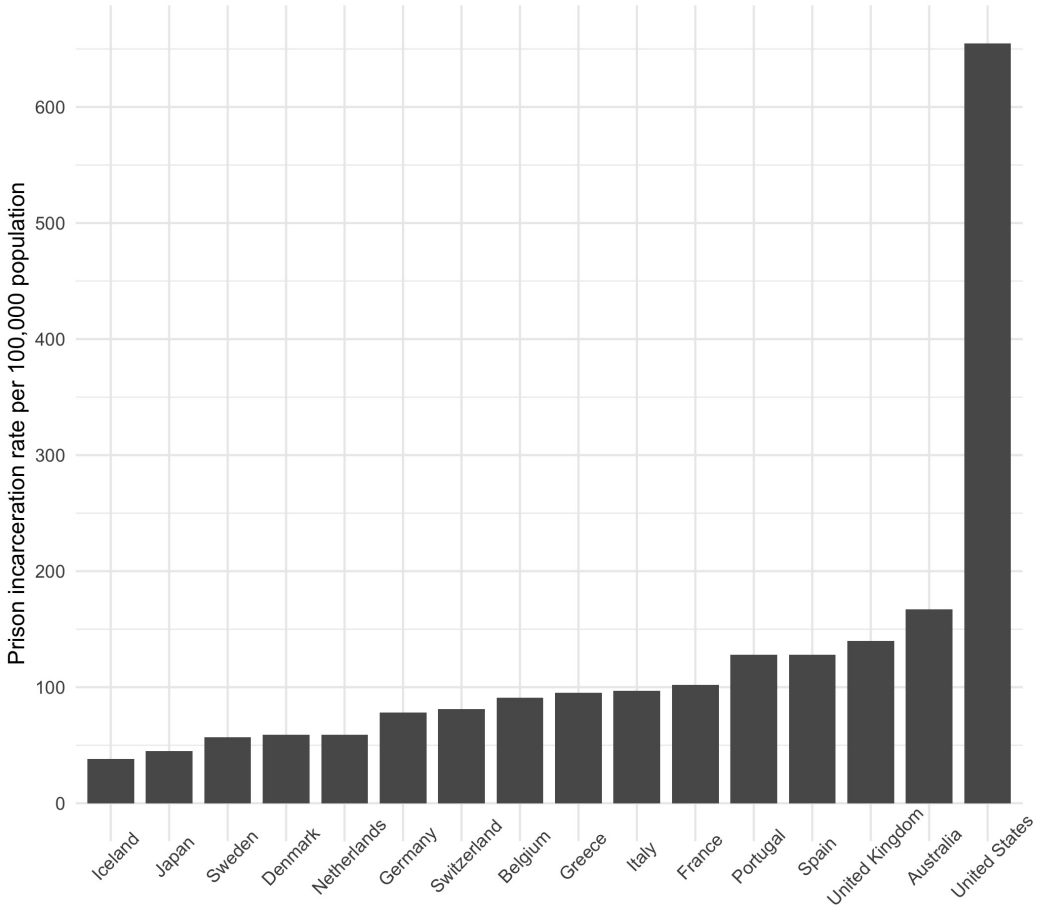

Figure 7: *Cross-national differences in prison incarceration rates between selected countries. Data source: World Prison Brief, Institute for Criminal Policy Research.*

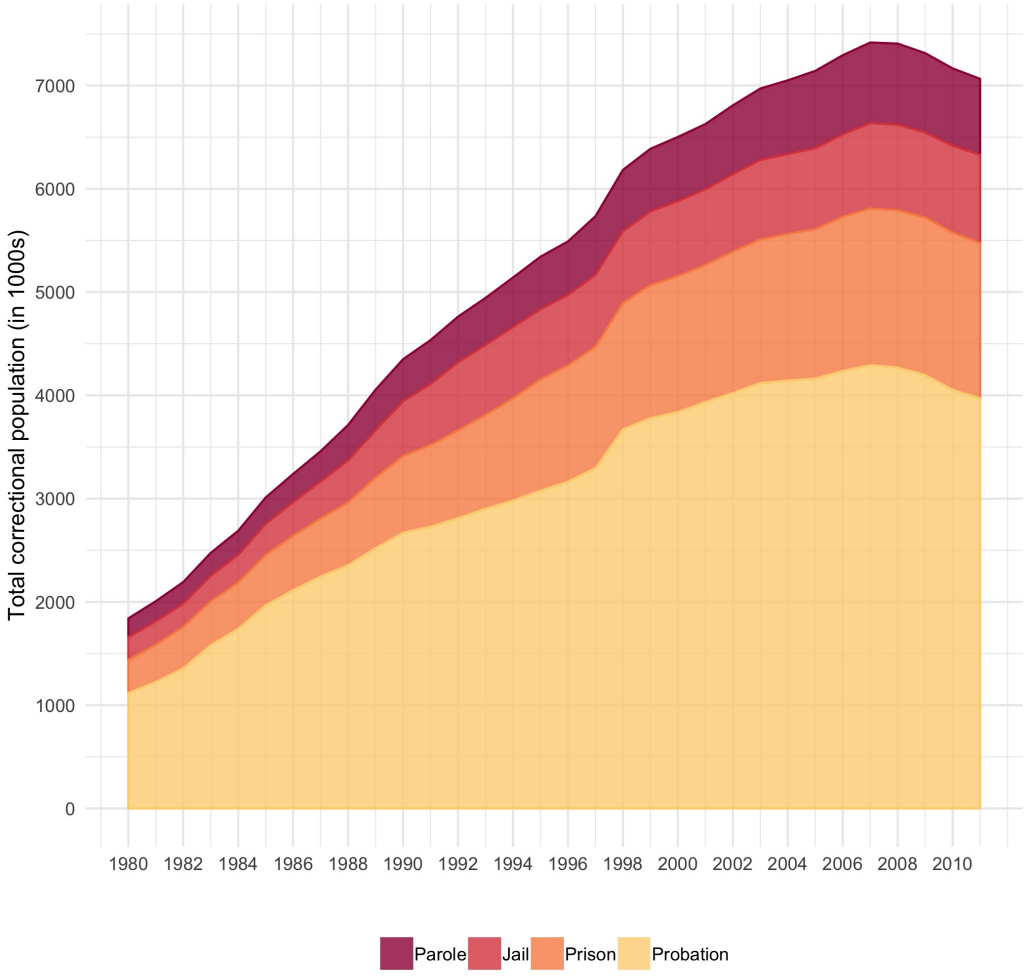

Figure 8: *Evolution of the American correctional population between 1980 and 2011. Data source: Sourcebook of Criminal Justice Statistics. See Figure 2-4 in National Research Council (2014: 41).*

Research Council, 2014). Rather, it was primarily the result of a series of sentencing reforms that included mandatory sentences for drug convictions and so-called "three strikes" and "truth in sentencing" laws (however, see Pfaff, 2017). These policies were themselves produced by a heightened focus on crime that reflected sensationalisation in the mass media and electoral considerations, in which Democrat and Republican candidates competed to be "tough on crime" (Beckett, 1997; Tonry, 1999; Smith, 2004). That there is a (causal) link between the size of America's correctional population and the magnitude of the ongoing overdose epidemic is a hypothesis that has received scarce attention at best. The objective of this thesis is to fill this gap, especially by relating the social structure of health inequality to the dynamics of American capitalism.

Thesis overview

The thesis is divided into two main parts. Part I is devoted, on the one hand, to conceptualising the phenomena under investigation, and, on the other hand, to historicising their emergence and ramifications. In chapter 1, I outline a theoretical framework that unifies seemingly disparate approaches to the study of health and illness. I advocate the analytic lens offered by the political economy of public health, which weds insights from various strands of social scientific research into a coherent and comprehensive mode of investigation. Chapter 2 retraces the historical dynamics of deindustrialisation and the political responses to subsequent socioeconomic convulsions that have led to the rise of the penal state, but also to a broader range of policy

mechanisms by which inequalities are produced and perpetuated. Chapter 3 outlines a series of hypotheses surrounding the causal pathways and mechanisms by which the interlocking dynamics of economic decline and punitive social policy can materialise as socially patterned distributions of disability, disease, and death, thus setting the stage for the subsequent analyses.

Part II comprises a series of empirical analyses. In chapter 4, I examine the association(s) between economic decline, jail and prison incarceration, and age-standardised mortality rates from drug use disorders at the U.S. county level between 1980 and 2014. Chapter 5 offers a second set of empirical analyses of broader inequalities in all-cause mortality rates, in premature mortality risk, and in life expectancy at birth at the county level between 1980 and 2014. Finally, in chapter 6, I assess the roles of deindustrialisation and prison incarceration rates in driving variation in life expectancy at age 40 in the top and bottom income quartiles at the state level between 2001 and 2014.

All the empirical chapters of which Part II is composed are modified versions of journal articles that are either currently under peer review or, in the case of chapter 6, have already been published (Nosrati et al., 2018). In all cases, I have designed the study, conducted the empirical analysis, and interpreted the results as lead author. I acknowledge the important contributions of my co-authors – Michael Ash, Jacob Kang-Brown, Lawrence King, Michael Marmot, and Martin McKee.[6]

[6] Listed alphabetically. Professor Lawrence King is the senior author on all papers.

Part I

Theory and history

Chapter 1

The social science of unequal life chances

1.1 Introduction

The sociological notion of "life chances" is derived from Max Weber's famous account of class situations, described as a shared set of probabilistically defined opportunity structures by which groups and individuals gain access to and appropriate social goods (Weber, 1978: 302, 927). That life chances vary systematically across social classes (and – to remain faithful to the Weberian idiom – across status groups) has in many ways constituted a starting point for sociological investigations of inequality. However, such investigations have tended, more often than not, to ignore that form of inequality of which the notion of *life* chances offers an almost literal echo, namely inequalities in life and death. In the present chapter, I seek to offer a conceptual basis for a social science of unequal life chances – one which spotlights dis-

parities in health and wellbeing, but as seen in tandem with (rather than in contradistinction to) the socially patterned distribution of material and symbolic goods. Moreover, I put a macroscopic emphasis on the upstream causal factors that generate and stratify probabilistically defined life course trajectories (cf. Beckfield, 2018) without forsaking the theoretical conception of the human animal as a sociospatially situated creature of flesh and blood (cf. Krieger, 2005; 2011; Wacquant, 2015). To achieve this, I proceed as follows. First, I offer an analytic definition of inequality and describe its three principal manifestations – namely resource inequality, existential inequality, and vital inequality (Therborn, 2013). Second, I outline three competing approaches to explaining vital inequality and explicate why I view the political economy of public health as the most potent. Finally, I unpack this latter framework by detailing its theoretical underpinnings, conceptual apparatus, and analytic assumptions, before outlining how it will be applied in the forthcoming analyses.

1.2 Inequality: forging an analytic concept

The concept of inequality has undergone a distinct transformation in recent times, passing from a state of pervasive neglect to one of widespread appropriation in public discourse, notably so after the publication of Thomas Piketty's mammoth tome on *Capital in the Twenty-First Century* (Piketty, 2014). Its buzzword-like diffusion has conferred upon it a somewhat hackneyed quality, wrought by a mix of overexposure and persistent ideological tension. For the social scientist, such a development has the twofold implica-

tion of raising public awareness of a crucial domain of social life and enquiry, but also of seeing an analytic category contract an enduring sense of indeterminacy. Not only does the emergent phraseology of "inequality" lend itself to varying discursive (ab)use but it can also serve to dilute the concept to the point that its internal rigour risks annulment. This is exemplified by the conflation of inequality and difference, or an assumed (but restrictive) equivalency between inequality and income distribution. Nonetheless, the concept does not need to be forsaken, provided it is analytically (re)constructed.

In what follows, I construe the concept of inequality as distinct from that of mere difference or multiplicity in that it captures and reflects the ways in which societally organised power governs the distribution of both material and symbolic goods. The most widely used definition of power – "the probability that one actor within a social relationship will be in a position to carry out [their] own will despite resistance" (Weber, 1978: 53) – stresses its distributive aspect, i.e. the power of A *over* B. As noted by Mann (2012), this can be complemented by a more functional aspect whereby power is manifest in the sociospatially embedded capacity for collective organisation. Both of these dimensions of power are present in Marx's emphasis on the interplay between "objectified" and "living" labour (Marx, 2000: 400), or, equivalently, in Bourdieu's emphasis on the capacity to "appropriate social energy" (Bourdieu, 1986: 241).[1] Inequality is thus an expression of *socially sanctioned disparities in human capabilities to function and flourish* (cf. Sen,

[1] Once again, the almost vitalist character of these phrases, echoing that of life chances, has remained conspicuously underexplored in sociological research. An interesting comparison can be made with the simple physical definition of power – the rate of doing work per unit time.

1995), anchored in macroscopic institutional configurations (such as state-crafting), bolstered by material infrastructures (the built environment), and durably inscribed into minds and bodies (the lived experience of symbolic categories).[2] It can be "soft" and latent, irretrievably lodged in the deepest infolds of the social unconscious, entombed in the immanence of a lifeworld (*Lebenswelt*), or it can be coercively enforced, via legitimate authority or – when push comes to shove – through the active deployment of concentrated lethal violence.

Following Göran Therborn, I differentiate between three forms of inequality. *Resource inequality* refers to the skewed distribution of income and wealth, but also of knowledge and cultural goods – i.e. what provides social agents with "unequal resources to act". *Existential inequality* is defined as "the unequal allocation of personhood, i.e., of autonomy, dignity, degrees of freedom, and of rights to respect and self-development". Finally, *vital inequality* – which forms the crux of the present plot – designates "socially constructed unequal life-chances of human organisms" (Therborn, 2013: 49).

[2] The reader may ask, why "sanctioned" (as compared to, say, "produced")? Inequality – especially what Charles Tilly (1999) dubbed "durable inequality" – is wrought by socially embedded and organisationally fashioned relations of power. Compliance with the established order is not necessarily located at the level of consciousness, but does, from time to time, need to be reinforced through collective organisation from above (one tends to forget that those at the top typically have a much richer, or at least more effective, "action repertoire" [Tilly, 2006] than those at the bottom). The victims of inequality need not always comply because they have "internalised" the status quo, but may simply do so, as Michael Mann puts it, "because they lack collective organization to do otherwise, because they are embedded within collective and distributive power organizations controlled by others. They are *organizationally outflanked*" (Mann, 2012: 7).

These three forms of inequality are irreducible but in no way mutually exclusive (*ibid*: 53, Table 3), and in my analysis I underscore how all three remain reciprocally mediated.

1.3 Three explanatory frameworks

In a famous passage in *The Strategy of Genes*, the biologist Conrad Waddington likens the process of cellular differentiation, whereby a cell transitions from a state of pluripotency to one of tissue-specific specialisation, to the trajectory of a marble rolling down an unevenly shaped hill (Waddington, 2014: 29). The pathway leading from the top to the bottom of the hill is mediated by a multilayered probability structure as defined by the troughs along the hillside, capable of tilting the marble's trajectory in one direction or another.[3] In a similar vein, Francis Galton demonstrated, already in 1889, that the passage of marbles (or, in his thought experiment, beans) through a "Quincunx" – a funnel containing a vertical board studded with strategically positioned pegs – is subject to a probabilistically defined destination structure that is largely independent of the individual marbles themselves (Galton,

[3] It is worth noting that Waddington's book was originally published only one year before John Gurdon's discovery that even fully specialised cells (that is, cells that are located somewhere at the very bottom of Waddington's landscape) contain all the genetic information of pluripotent cells (i.e. cells located near the top of the hill) (Gurdon et al., 1958). This foreshadowed the demonstration, decades later, that under the right circumstances, pluripotency can be induced by effacing the epigenetic signature fostered by cellular differentiation (Takahashi and Yamanaka, 2006). In other words, a given destination structure is neither inevitable nor necessarily irreversible.

1889). Both Galton's "Quincunx" and Waddington's "epigenetic landscape" offer helpful metaphors for the social science of unequal life chances (cf. Krieger, 2012). They evoke the simple question of what determines these structured pathways to begin with: how come some marbles end up at point A rather than at point B (or C or D or E, etc.)? Put differently, why are resources, status, and longevity unequally distributed across social and physical space? What generates the systematic patterning of human trajectories? In statistical jargon, what is the "data generating process"? And, more specific to this particular research, how can persistent disparities in health and wellbeing be scientifically explained?

For lack of offering an exhaustive account of diverse approaches to the study of vital inequality,[4] I will confine this conceptual sketch to a distinction between three principal explanatory frameworks. The first, which currently dominates the fields of medicine, public health, and epidemiology (McMichael, 1999), is one that emphasises the proximal determinants of health (see Birn et al., 2017: 90–92). This framework encompasses two main internal models. The *biomedical model* is characterised by three distinctive features: (*a*) it restricts its attention to biological, chemical, and physical phenomena; (*b*) it espouses a methodological approach that privileges (randomised) clinical trials or, more generally, a potential outcomes conception of causality;[5] and (*c*) it tends to adopt the view that phenomena are best

[4] For an extensive overview of different varieties of epidemiological theory, their histories, and their interconnections, see Krieger (2011).

[5] See Krieger and Davey Smith (2016) and accompanying comments in a special issue of the *International Journal of Epidemiology* for a critical discussion of epidemiological conceptualisations of causality.

explained by the properties of their parts (Krieger, 2011: 130). Despite its drive to identify previously unknown pathogenic biological mechanisms and to promote large-scale investments in a range of clinical (especially pharmacological) interventions, this model is entirely bereft of the capacity to explain population-level inequalities in health, other than via recourse to an aggregated description of differences in biological make-up between individuals. Its methodological individualism is oriented towards the dissociation of discrete units of analysis – that is, individuals composed more or less uniquely of biological, chemical, or physical traits – from their broader ecological habitat. The *lifestyle model*, on the other hand, relies on a perspective according to which unequal exposure to "risk factors" is a product of differences in behaviour, especially consumer behaviours related to smoking, physical exercise, and diet (*ibid*: chapter 5, especially pages 140–148). To account for the ongoing overdose epidemic, for instance, this framework highlights the supply of opioids and other drugs, coupled with the "choices" or "incentives" to consume them, as key determinants of inequality. Whilst this approach has the merit of tracking micro-level behaviours that are detrimental to population health, it fails – much like the biomedical approach – to relate such behaviours to structural constraints that lie beyond the scope of the individual (or groups of aggregated individuals) and falls short of explaining how such behaviours are generated in the first place, why they remain socially patterned, or why they are subject to substantial variation across time and space. In short, this framework has limited, if any, capacity to unpack the layered pathways of Waddington's landscape or Galton's board.

The second framework is that of social epidemiology, which takes an im-

portant step in the direction of providing a more relational understanding of health and illness. As opposed to the biomedical or lifestyle approach, it highlights the importance of social context, it posits that social phenomena can causally impact distributions of health and disease, and it recognises that population health is a dynamic, not a static phenomenon (*ibid*: chapter 6). Chief amongst the social determinants of health are factors like poverty, unemployment, income inequality, racial discrimination, or neighbourhood context (Marmot, 2005; Berkman et al., 2014). This strand of research has experienced continued success in recent years, and has shed crucial light on how social, behavioural, and biological phenomena must be viewed as pathogenically integrated. Moreover, it arguably offers the first conceptually and empirically coherent explanation for the existence (and persistence) of social inequalities in life and death. However, although it moves further up the causal chain than do the biomedical and lifestyle models, it rarely examines *the social determinants of the social determinants of health*. As argued by Anne-Emmanuelle Birn, social epidemiology claims to examine "the causes of the causes" of ill health and inequality, but remains conspicuously silent when it come to the *causes of "the causes of the causes"* (Birn, 2009). In other words, it rarely asks why factors like poverty, unemployment, or discrimination materialise in the first place. To revisit our metaphor, it recognises that marbles can follow different – socially mediated – pathways but asks few questions about the (political and economic) roots of existing population distributions and destination structures.

The third and final framework, which is the one adopted in this thesis, is that of the political economy of public health. This is an emergent (some

would say re-emergent) research stream that seeks to understand the distal social, economic, and political causes of population health (cf. Doyal, 1979; McKinlay, 1979). What distinguishes it from the previous two frameworks is not only its macroscopic lens, geared towards unpacking large-scale phenomena that fashion or fissure, design or disrupt entire societies, but its primordial attention to the societal organisation of power (see Birn et al., 2017: 101–103). This is ultimately what renders it capable of accounting for the making, remaking, or unmaking of (vital) inequality. It thus probes the *societal* (not just social) determinants of ill health and locates existing social phenomena in the context of broader historical and political dynamics (Krieger, 2011: 185; Birn et al., 2017: chapter 3; see also Krieger and Beckfield, 2009). In short, it retraces the *structural properties* that determine the distributional features of Waddington's landscape or Galton's "Quincunx" (Beckfield, 2018). Examples of such an approach include studies of the effects of mass privatisation policies in driving the post-communist mortality crisis (Stuckler et al., 2009); the impact of austerity policies on mental health in Europe (Reeves et al., 2016); the role of corporations in shaping unhealthy behaviour like smoking and unhealthy food and drink consumption (Stuckler et al., 2012); the structural connections between work(lessness) and health inequality (Bambra, 2011); or the profound effects of institutionalised racism – in the form of Jim Crow laws – on inequities in infant mortality rates across American states (Krieger et al., 2013). This constitutes a return to the origins of public health, captured by Rudolph Virchow's famous dictum: "Medicine is a social science, and politics is nothing more than medicine on a grand scale" (Virchow, 1848).

1.4 Putting the political economy of public health to work

My usage of this framework rests on three analytic pillars. First, I construe current social, political, and economic forces are layered sediments of the past. "The tradition of all the dead generations weighs like a nightmare on the brain of the living", as Marx (2000: 454) famously said in 1852. In the 1867 preface to *Das Kapital*, he adds that "[we] suffer not only from the living, but from the dead. *Le mort saisit le vif* [the dead man clutches onto the living]!" (Marx, 1976: 91). Insofar as social science may be defined as a "history of the present" (to paraphrase Foucault [1995]), I will therefore seek to historicise the determinants of unequal life chances. This will be the principal focus of chapter 2, where I retrace the emergence and ramifications of deindustrialisation, penal expansion, and punitive social policy in the American context.

Second, insofar as this framework pays particular heed to the societal organisation of power, I posit that the emergence, morphology, and dynamics of (vital) inequality must be explicitly related to the logics of capitalism (Marx, 1976). Capitalism, as a distinctive historical phenomenon based on institutionalised relations of power, can (for present purposes) be defined as a system of generalised commodity production characterised by (*a*) the private ownership of the major means of production, (*b*) formally free labour, and (*c*) the invention of credit-money, all under the aegis of a strong bureaucratic state.[6] This entails an emphasis on what Weber dubbed the "memorable al-

[6] For discussions of alternative definitions, see e.g. Ingham (2011), Kocka (2016), or

liance" between the state and a relatively autonomous economic elite (Weber, 1978: 353) through which inequalities between labour and capital are embedded in the social relations of production and ratified through the deployment of bureaucratic rationality. Different modalities of this very "alliance" and its political and economic consequences are central to understanding the roots of rapid industrial decline and the crafting of social policy in the United States, as will be described in the next chapter.

Third (and here I complete my recourse to sociology's foundational insights, this time by drawing on Durkheim), I spotlight the social alchemy whereby symbolic representations – and their dissolution – morph into enduring material realities that shape the trajectories of individuals and collectives alike (Durkheim, 2002; 2008). Insofar as institutions (from labour markets to prisons) can be construed as "realised categories" (Bourdieu, 1993a), i.e. as materialisations of social principles of vision and division, they organise power not only through the distribution of tangible resources but also through the enforcement of symbolic schemas (Clemens and Cook, 1999; Beckfield, 2018: 17–23). The focus on macro-level institutional arrangements enabled by the political economy of public health thus implies an engagement with the (re)production of categories and classifications that prove consequential for the making of life chances, including (for instance) those involved in ethno-racial formation or embodied stigma and social relegation.

How do these analytic and conceptual foundations map onto my applica-

Hodgson (2016). For a rich discussion of American capitalism in light of recent historical scholarship, see Beckert and Rockman (2016) and Beckert and Desan (2018). For a broader historical (and historiographical) account, see Kocka and van der Linden (2016).

tion of the political economy of public health in the present study? In the forthcoming chapters, I seek to explain the state of America's population health landscape with reference to the ripple effects of socioeconomic disruption – notably in the form of industrial decline – and the political responses to such developments, especially in the guise of high rates of incarceration. On the one hand, I view deindustrialisation as a historically distinct manifestation of "creative destruction" (to borrow Schumpeter's [1994] evocative phrase) propelled by the dynamics, both internal and global, of American capitalism. On the other hand, the historically unprecedented expansion of the penal apparatus is construed as part of a broader public policy repertoire through which attendant social divisions are curbed and controlled, managed and magnified. The potency of the political economy approach is easily identified when considering that spatial variation in life chances across the United States is politically and economically driven, conveyed by the consequential legacies of economic decline in Appalachia and the Rust Belt on the one hand, and of slavery and institutionalised racial domination in the South on the other, not to mention that within-U.S. variation is itself politically rooted in the institution(s) of federalism (Beckfield, 2018: 77, 79; see also Kunitz, 2015).

At the intersection of these macro-level developments is the (welfare) state. The latter may be construed as a wide-ranging stratification machine which operates, in the words of Gösta Esping-Andersen, "not just [as] a mechanism that intervenes in, and possibly corrects, the structure of inequality; it is, in its own right, a system of stratification. It is an active force in the ordering of social relations" (Esping-Andersen, 1989: 23). By virtue of its

monopoly on the legitimate exercise of physical and symbolic violence (Bourdieu, 1993b; Weber, 2004), the state forms the locus of social struggle for the division between "insiders" and "outsiders", between those who "belong" and those who do not, between the "deserving" and the "underserving", and it thus actively fabricates the subjective and objective conditions undergirding unequal life chances. Incarceration is a primal example of this power. The prison is a politically rooted institution that actively stratifies a population, delineates and aligns group boundaries, and thus inscribes symbolic divisions into materiality by institutionalising social categories (Shannon and Uggen, 2012). In a word, the prison is a pivot of exclusionary closure and thus an upstream determinant of inequality.

Social policy is typically seen as encompassing a set of government interventions in the realms of employment, pensions, housing, and health care – but not criminal justice. However, the conceptualisation of penal policy as social policy can be motivated on at least two levels. At the historical level, it is worth recalling that the initial conception of the prison in late sixteenth-century Europe, before ever being construed as a unique tool of punishment or rehabilitation, was as "an instrument of social policy with regard to beggars" (Geremek, 1994: 207), geared towards the regulation and invisibilisation of poverty at the dawn of modern capitalism. A means of warehousing landless vagrants uprooted by the enclosure movement and of curbing the social convulsions wrought by the commodification of labour, the penal wing of the (welfare) state thus historically preceded the currently more familiar social wing that emerged in the late nineteenth century under the press of industrialisation. Moreover, the social wing itself has not

only been oriented towards providing security for society's most vulnerable but, historically, has derived from an active conservative effort to quell the revolutionary leanings of the early labour movement and to sanctify loyalty to the state by managing some of the most deleterious effects of industrial capitalism at the bottom of the class structure, as evidenced by the rise of welfare policies under Napoleon III in France, under Bismarck in Germany, or under von Taaffe in Austria (see Esping-Andersen, 1989: chapter 2; see also Piven and Cloward, 1993). From this it may be argued that, at the analytic level, the dissociation of penal policy with social policy is little but a symptom of disciplinary sectarianism. Bringing social policy and criminal justice together conceptually within a single framework not only dissolves an arbitrary boundary between mutually imbricated objects of empirical enquiry but also facilitates a broader, unifying understanding of the interconnections between different flanks of the same political economy of inequality (Beckett and Western, 2001; Wacquant, 2009). Such a unifying endeavour is one of the principal objectives of the present research.

By construing incarceration as social policy, I draw on existing empirical and theoretical research on the role of the welfare state in the making of vital inequality (see Bambra, 2007; Eikemo and Bambra, 2008; Lundberg et al., 2008; Beckfield et al., 2015; Beckfield and Bambra, 2016). However, following Loïc Wacquant, I also view the penal state as embedded in a broader reconfiguration of the "memorable alliance" between state and capital, geared towards punitive interventionism at the bottom of the American class structure and protective guardianship at the top (Wacquant, 2009; 2010). As I will expound in the next chapter, this entails the retrenchment of social assistance

for the most vulnerable, the reorienting of welfare towards workfare, and the expansion of the penal system; but it also entails a generous welfare system for the wealthy (Faricy, 2016), coupled with the re-regulation of broader institutional dynamics in favour of corporations (Domhoff, 2014). Drawing on the work of Jason Beckfield and colleagues, I conceptually designate this phenomenon as *institutional imbrication*, which refers to the "simultaneous operation of institutions in multiple domains at multiple levels" (Beckfield et al., 2015: 233), notably through the "[combination of] multiple policy exposures and [channelling of] their overlapping effects to variable parts of the population" (Beckfield, 2018: 121). Crucially, this concept "allows for amplifying, cross-cutting or moderating effects of institutional arrangements, accurately reflecting the reality that people 'live' more than one policy at a time over the life course" (*ibid*: 126).

1.5 Conclusion

In this chapter, I have argued that inequality is an expression of socially sanctioned disparities in human capabilities to function and flourish that manifest as resource inequality, existential inequality, and vital inequality. Further, I have argued that the political economy of public health offers a unique framework through which the upstream determinants of vital inequality can be grasped. It is distinct from other predominant frameworks in its attention to the societal organisation of power through which unequal life chances are produced and reproduced. It historicises overlapping patterns of inequality and retraces their embeddedness in macro-level institutional configurations

that govern the distribution of material and symbolic goods. In seeking to shed novel light on the roots of deepening vital inequality in the United States, I have emphasised the need to study the structure and dynamics of capitalism, with a special focus on the operations of the (welfare) state in the wake of rapid socioeconomic change. This will be the central focus of the next chapter.

Chapter 2

(Vital) inequality and the dynamics of capitalism

2.1 Introduction

The purpose of this chapter is to situate my object of analysis in a historically anchored set of developments of American capitalism. I begin by sketching the history of industrial decline in the post-World War II period, which has been at the centre of public and academic debates surrounding economic change and inequality at the dawn of the 21st century. I will argue that key features of deindustrialisation, notably the reallocation of investment across economic sectors and the selective reterritorialisation of economic activity, must be related to the logics of capital and its "conceptions of control" (cf. Fligstein, 1990). I will describe the ripple effects of deindustrialisation on the social patterning of (un)employment relations, concentrated poverty, and social relegation. In a second step, I will examine the parallel expansion of

the American penal state since the early 1970s and detail its core institutional properties. Finally, I will connect penal policy to social policy more broadly, and will examine the socially differentiated structure of the American welfare state, which operates in deeply contrasting ways across different layers of the class structure.

2.2 The political economy of "creative destruction"

Three empirical facts surrounding American deindustrialisation stand out. First, as a share of total national non-farm employment, manufacturing employment has undergone a continual decline in recent decades, falling from 32% in 1948 to 8% in 2017. The number of workers employed in the manufacturing sector reached a peak in 1979, counting over 19 million, and hit its lowest level mid-recession in 2010, with just over 11 million. However, the net manufacturing employment change of -6.6 million between 1977 and 2012 masks additional gross flows that have caused major dislocations within the labour force. Second, manufacturing employment dropped by 12% between 1979 and 2000, then plummeted by another 25% between 2000 and 2012. And third, this decline has unfurled in parallel with a steady increase in labour productivity in the manufacturing sector over the same period, as evidenced by rising real output and close to $2.5 trillion real value added just before the 2009 recession, compared to around $0.5 trillion in the late 1950s (Fort et al., 2018; U.S. Bureau of Labor Statistics, n.d.). The root causes of these developments are contested (see e.g. Rodwin and Sazanami, 1989;

Alderson, 1999; Brady and Denniston, 2006), but the extant literature converges around two explanatory factors, namely globalisation-induced trade competition and technological change.[1]

A number of econometric studies suggest that Chinese import penetration may have accounted for one quarter – or, in some model specifications, up to one half – of the overall employment decline in manufacturing since 2000 (Autor et al., 2013; see also Bernard et al., 2006; Autor et al., 2014; Acemoglu et al., 2015; Pierce and Schott, 2016a). It is worth highlighting the difference between *direct imports*, i.e. finished goods, and *import penetration*, which involves heterogeneous corporate sourcing decisions across domestic and foreign supply chains. These two modalities of economic activity seem to have divergent effects on employment patterns, with the latter having a *stronger* association with job destruction in manufacturing than the former (Antràs et al., 2017). Other studies have tried to isolate exogenous variation in automation, finding that the adoption of industrial robots or the computerisation of routine tasks precipitate further employment declines (Autor et al., 2015; Collard-Wexler and De Loecker, 2015; Acemoglu and Restrepo, 2017). However, the overall trend has been one of simultaneous rises in import penetration and technology adoption, rendering the forced disentangling of the two processes a potentially futile endeavour. Existing evidence shows that the rate of technology adoption as a share of firms in the manufacturing sector underwent a steady increase at the tail end of the previous century, before jumping up around the time of establishing Permanent

[1] Notwithstanding those who view deindustrialisation as an inevitable and natural consequence of economic development (e.g. Rowthorn and Ramaswamy, 1997).

Normal Trade Relations to China in October 2000. Computerisation within American plants grew from less than 5% of all firms in 1977 to over 20% in 2000, at which point it galloped to reach over 60% in 2002, following which it declined somewhat but remained at a high level (over 40%). At the same time, import penetration grew steadily from less than 10% in 1977 to just over 20% in 2000, and to around 30% by 2012 (Fort et al., 2018: 52, Figure 2). Indeed, it may be that technological change is itself a response to trade shocks (*ibid*: 54; cf. Acemoglu, 2002; Oldenski, 2014).

Insofar as the principal dispute in the economics literature surrounding deindustrialisation focuses on the relative explanatory capacity of trade versus technology, the question of (corporate) power has been largely neglected in mainstream accounts. Automation and other technological change may not only be endogenous to trade liberalisation, but both developments are embedded in broader power dynamics, as evidenced by how the designs of trade agreements typically reflect the interests of politically well-connected firms, such as international banks, pharmaceutical companies, and other multinational corporations (for a recent discussion, see Rodrik, 2018). Moreover, as the above figures suggest, trade competition from low-wage countries and attendant technological changes involved in the production process only account for a given portion of degrading employment parameters and wage stagnation or decline amongst workers. Most importantly, these developments alone do not seem to account for the increased share of surplus appropriated by capital. In 2014, the share of national income earned by the top 1% of adults (approximately 2 million individuals) in the United States equalled around 20%, compared to only 13% for the bottom 50% of the adult

population (approximately 117 million individuals), marking an almost exact reversal since the mid-1970s, when the corresponding figures were around 11% and 20%, respectively. Thus virtually the entire 60% increase in average pre-tax real national income per adult since 1980 has gone to the top income earners, who enjoyed an average income of over $1.3 million in 2014, whilst income in the bottom half of the population has stagnated at around $16,500 per annum (one-fourth of the average national income), with the modest post-tax income growth in this group being absorbed by increased health spending (Piketty et al., 2018). This has been coupled with tripled wealth concentration amongst the top 0.1% wealth holders, from 7% of total national wealth in 1978 to 22% in 2012, and a sharply declining wealth share in the bottom 90%, from a peak of over 35% in the mid-1980s to around 22% in 2012, as a result of sapped savings and burgeoning mortgage, consumer, and student debts (Saez and Zucman, 2016; see also Alvaredo et al., 2018).

These patterns of resource inequality can be related to those of industrial decline. The distributional impacts of trade liberalisation have principally manifested as occupational polarisation and wage repression amongst low-educated, low-skill workers, especially for those working in industries that were disproportionately affected by tariff reductions under the North Atlantic Free Trade Agreement. A comparison of the manufacturing sector with unaffected industries suggests a drop in wage growth of 17 percentage points associated with trade shocks between 1990 and 2000 (Hakobyan and McLaren, 2016; see also Oldenski, 2014). Statistically robust but smaller adverse impacts have been associated with technological change across both employment and wage parameters (Acemoglu and Restrepo, 2017; see also

Autor et al., 2015; Graetz and Michaels, 2017). Meanwhile, overall corporate profits have increased sharply, standing at over $1.9 trillion (after tax) in 2014. In the manufacturing sector, employment declines are overshadowed by rapid value-added growth, with corporate profits reaching close to $500 billion in the same year (U.S. Bureau of Economic Analysis, n.d.).

The *political* economy of "creative destruction" can be gleaned from three significant structural patterns. First, it is noteworthy that net firm death accounts for no more than a quarter of overall industrial decline between 1977 and 2012, and variation across establishments nested within firms in continuous operation accounts for over 60% of the overall employment change (Fort et al., 2018: 50, 59–60, Figure 4). What is at stake is not the evaporation but the reallocation of capital, which can be achieved by redirecting profits from one site of production to new facilities or other activities; by failing to renew factors of production and maintaining a site of production; by selling off a plant's capital stock via subcontracting or outsourcing; or, ultimately, by plant closure and complete relocation of production via offshoring (Bluestone and Harrison, 1982: 7). However, such capital mobility relies on complex institutional configurations that allow corporations to combine the spatial dispersal of production with the economic concentration of ownership in their pursuit of compound accumulation and growth.[2] The

[2] Contrary to discourses linking globalisation to the evaporation of states and their borders, most available evidence suggests that assemblages of global operation, coordination, and control are mediated by the state as the ultimate guarantor of the rights of capital through the fabrication and enforcement of property rights, the protection of contracts, the mobilisation of labour power, and the territorialisation of production processes (e.g. Fligstein, 2001; Sassen, 2008; Panitch and Gindin, 2013). The state thus emerges as a core

pursuit of profitability results in distinctly patterned capital shifts whereby subsidiaries are bought and sold according to strategies designed to optimise corporate portfolios (cf. Cowie, 2001).[3] In the process, a drive for diversification has propped up firm profits whilst leaving abandoned industries and their production sites hollowed out. This drive intensified in the 1970s and continued well beyond, as employment levels in non-manufacturing establishments owned by manufacturing firms grew steadily until around 2000, at which point they plateaued (Fort et al., 2018: 67, Figure 7). The geography of such capital mobility can be divided into a first stage, before the year 2000, during which the dominant trend was one of "domestic offshoring" as seen in consistent employment reallocation away from organised labour in the industrial heartland towards non-unionised and "pro-business" state regions (Holmes, 1998; Bernard et al., 2013; Fort, 2017), until a second stage facilitated the shifting of operations to low-wage foreign countries after the

supplier of institutional infrastructures suited to harnessing market dynamics in favour of capital (cf. Campbell and Lindberg, 1990).

[3] In their famous account of the early waves of deindustrialisation in the United States, Bluestone and Harrison report that four categories emerged from the arraying of a product line's market share against the sales growth rate of each corporate activity: (*i*) lines with both low market shares and growth rates; (*ii*) lines with high market shares but low growth rates; (*iii*) lines with still low market shares but rapid growth rates; and (*iv*) high-share, high-growth "star" lines. The codification offered in the 1970s by the Boston Consulting Group, a distinguished management consulting firm, recommended completely abandoning the first category (which failed to meet minimum profit targets, sometimes set at no less than 25%), "milking" the second until a limit had been reached before shifting investment to the third, whilst adopting a provisional *laissez-faire* attitude towards the fourth (Bluestone and Harrison, 1982: 150–152).

turn of the millennium.

Second, the drive for diversification has been accompanied by mergers and acquisitions that contribute to oligopolistic concentrations of capital. From the early years of industrialisation (cf. Beckert, 2003), this issued in a mode of industrial organisation whereby absentee control of establishments, i.e. remote management of production sites by central headquarters external to and hence detached from local economies, allowed corporate operations to cut across various social and spatial divides and further facilitated the type of flexibility associated with rapid capital shifts, divestiture, and reinvestment (Bluestone and Harrison, 1982: 157; cf. Caves, 1980; Aaron, 1983; Staudohar and Brown, 1987). Large corporations and conglomerates have been disproportionately responsible for job destruction rates, and closures have been more likely to occur in multinational, multi-plant firms (Bernard and Jensen, 2002).

Third, the spatially differentiated drive to expand and contract has motivated the strategic erosion of organised labour in geographies where union activism initially emerged, namely areas characterised by strong industrial histories. The shifting of operations to the Sun Belt, to the non-unionised peripheries of the North and the West, and to foreign establishments encompasses two prominent strategies. On the one hand, parallel production lines can serve to undermine union shops via duplicate production facilities in non-unionised geographies, allowing management to redirect production to the latter in the case of strikes or other forms of turbulence in the former. On the other hand, multiple sourcing allows corporations to pit smaller suppliers of components against one another by refusing to grant sole source

arrangements to independent shops. This weakens local supplier production runs and saps union density in supplier shops (Bluestone and Harrison, 1982: 166–167; see also Cowie, 1999; Lee, 2005).

On the level of legislation, the corporate community has developed an interlocking web of organisational resources that actively shape government policy outcomes (Domhoff, 2014; Gilens, 2014). From the 1935 National Labor Relations (or Wagner) Act, which legalised organised unions and conferred bargaining power over wages and work rules to their members, to the 1938 Fair Labor Standards Act, which inscribed minimum wages, the eight-hour day, and the abolition of child labour in the wage-labour compact, the labour movement was gaining ground in the pre-World War II economy (see e.g. Dubofsky and McCartin, 2017). The social conquests of the New Deal Era were compromised by the 1947 Taft-Hartley Act, which undermined union density and reoriented the social contract between labour and capital towards a workfare regime under the banner of "right-to-work" laws. The effectiveness of this legislative shift is illustrated by a rapid drop in levels of unionisation in the United States since 1947: after reaching a historical high of close to 35% following the National Labor Relations Act in 1935, there has been a steady decline in the percentage of unionised workers in the years following World War II until the present day (Carter et al., 2006). Between 1973 and 2007, private sector union membership plummeted from 34% to 8% for men and from 16% to 6% for women, whilst wage inequality grew by over 40% (Western and Rosenfeld, 2011; cf. Mishel et al., 2012; see also Denice and Rosenfeld, 2018). Similarly, the frequency of strikes and successful union elections has plummeted drastically (Carter et al., 2006), whilst the

real value of the median and minimum wages, as well as total earnings for a full-time minimum wage job as a share of poverty earnings, have dwindled since the late 1960s (Massey, 2008: 167–168).

Perhaps the most incisive sociological portrait of the early effects of deindustrialisation has been furnished by William Julius Wilson's study of *The Truly Disadvantaged* (2012). Wilson chronicles the rapid social and economic decomposition of the Black urban metropolis in the wake of industrial restructuring by retracing the conjoint rise of pervasive joblessness, violent crime, and family disintegration. Starting in the 1960s, what was previously the receptacle of industrial labour power, harbouring a disproportionate number of African Americans employed in blue-collar occupations, underwent an astounding implosion whereby the urban ghetto was transformed from a dual institutional device of ethno-racial seclusion and economic extraction into a functionally obsolete container of social stigma, concentrated poverty, and collective disarray (see also Wilson, 1997).[4] Economic dislocation spawned an increasingly dualised occupational structure that heightened insecurity at the bottom of the class spectrum, where low-skill, low-educated African Americans were mired in a situation of rampant unemployment punctuated by precarious wage work. What Wilson dubs "concentration effects", by which he designates the sociospatially distinct cumulative experience of relegation, were amplified by the rapid exodus of the Black middle class from the collapsing ghetto, depriving those left behind of a social bulwark against continued deprivation.

[4] To compare the "communal" industrial ghetto to the deindustrialised "hyperghetto", see Drake and Cayton (2015) for the former and Wacquant (2008) for the latter.

Whereas the early waves of industrial decline disproportionately impacted African Americans (cf. Bound and Holzer, 2000), the continued deproletarianisation of the American working class has had spillover effects across ethno-racial boundaries (see Sugrue, 1996; Neel, 2018). Narrowing Black-White employment ratios seem to be driven by increased within-category differentiation. Over and above the effects of increased school enrolment, labour force participation rates have declined by 3.2 percentage points amongst prime-age Whites since 2000 (Hipple, 2016), and are falling precipitously amongst those with no higher education (Case and Deaton, 2017). Overall labour force participation rates amongst men aged 25 to 54 without college degrees have declined by 6.4 percentage points between 1995 and 2015, and by 5.9 percentage points since 2000 (Hipple, 2016), dropping below 85% after the 2008 recession (Krueger, 2017). Annual volatility around average earnings amongst male workers doubled from 1974 to 2000 (Gottschalk and Moffitt, 2009), and seems to have continued into the new century. Labour market segmentation, union dissolution, and wage repression have fuelled this development (Abraham and Kearny, 2018; Denice and Rosenfeld, 2018; see also Western et al., 2012; Desmond and Western, 2018).

2.3 The rise of the penal state

The political response to the social and economic convulsions outlined above has been largely punitive. As described in the Introduction, the American penal state has undergone an expansion that is unparalleled, and it is the sociospatially concentrated human remnants of large-scale economic degra-

dation that form the principal clientele of the criminal justice system. To assess the causes of rising incarceration rates, it is helpful to decompose the main features of a growing prison population. The size of a prison population can be thought of as a function of crime rates, the probability of arrest given a crime, the probability of prison admission given an arrest, and the duration of prison sentences (National Research Council, 2014: 47). What configuration of these four elements has fostered the expansion of the penal state in recent decades?

There is no unilinear connection between incarceration and crime rates. In tandem with early waves of industrial decline, crime rates increased between the early 1960s and 1980s, as did incarceration rates (from 1973 onwards). However, whilst crime rates underwent a significant downswing in the following two decades, especially from the early 1990s, incarceration rates continued their strong upwards trajectory. This increase was seen across all crime categories but especially for drug-related crime. Imprisonment rates for drug offences jumped from 15 to 143 per 100,000 population between 1980 and 2010 (Beck and Blumenstein, 2012). Arrest rates per offences remained largely stable between 1980 and 2010, although arrests for murder declined by some 20% after year 2000 and arrests for aggravated assault grew marginally before 2000. In contrast, drug law enforcement efforts manifested in the form of rapidly rising arrest rates for drug possession and use, reaching a peak in 2006 at 162% above the 1980 level (National Research Council, 2014: 50). Prison admissions rates per arrests also increased, mostly through commitments related to murder offences – which grew from around 40 to over 90 prison commitments per 100 adult arrests – but also through assault and drug

offences. Prison admissions for the latter category grew rapidly throughout the 1980s but then flatlined after the early 1990s at around 10 prison commitments per 100 adult arrests (Beck and Blumenstein, 2012). Finally, as for time served in prison, conservative estimates suggest that sentence duration grew most dramatically for murder – by 238% between 1981 and 2000 – followed by sexual assault, which almost doubled between 1981 and 2009. Interestingly, the smallest sentence duration increase was for drug offences, which never exceeded 2 years, growing from 1.6 to 1.9 years between 1981 and 2000 and remaining stable at that level into the new century (*ibid*).

Overall, a decomposition of prison growth between 1980 and 2010 suggests that changing prison commitments per arrest coupled with longer sentence duration account for the bulk of the total variation (*ibid*). This largely corresponds to stricter enforcement for drug-related crimes under the "war on drugs" on the one hand, starting in the 1970s under President Nixon, and the subsequent wave of legislative moves to expand sentences. The advent of new sentencing policies unfolded from the mid-1980s to the mid-1990s, during which time a series of reforms were introduced to make sentences not only harsher but also more consistently imposed for crimes related to drugs and violence. This aim was primarily to be reached via mandatory minimum sentences, "three strikes" laws, "truth in sentencing" laws, and laws introducing the abrogation of parole for certain offences (see National Research Council, 2014: 71–85). As suggested by the above figures, those who serve the longer sentences are primarily under lock and key for violent offences, notably murder and assault, even as murder arrest rates have declined at century's dawn. Conversely, aggressive law enforcement for drug-related crimes have trans-

lated into significantly higher admissions rates with relatively short sentences (*ibid*: 54). This is reflected in how prisoners doing time for drug offences make up about one-fifth of the total carceral population at any one point in time, and prisoners doing time for non-violent drug offences make up only 6% of the total (Pfaff, 2017). However, the dramatic rise of drug-related arrests has translated into high turnover rates for minor offences, notably via local county jails rather than state prisons. In 2014, state and federal prisons held 1,562,300 people, usually convicted of felonies with a sentence of 1 year or more, whereas jails held 744,600 people who were predominantly in pre-trial detention. Whilst at any point in time jails held about half as many people as prisons, there were 11.4 million annual admissions to jails – almost twenty times the 626,096 people newly admitted to prisons each year.

Caution is therefore warranted in interpreting statistics that are typically presented in the debate surrounding "mass incarceration". The rise of the penal state must also be viewed against the historical backdrop of successive institutional forces by which the nexus of class and race has been shaped throughout American history, from the institution of slavery to that of incarceration via the Jim Crow system in the postbellum South and the Black ghetto in the industrial North (see Wacquant, 2009: chapter 6; Muller, 2012; 2018). For instance, despite the fact that, based on virtually all existing sources of evidence, rates of cocaine, crack cocaine, and heroin use amongst Whites consistently surpass those of Blacks (National Research Council, 2014: 50), the nominal share of drug crimes amongst African Americans jumped from around 30% to over 45% in the 1980s alone (Alexander, 2012). Equally telling is how law enforcement agencies have primarily tar-

geted activities such as public urination, rough sleeping, or begging for food (Thompson, 2010: 712), which is revelatory of a *punitive treatment of poverty* rather than crime, fiercely levelled at select spaces of socioeconomic relegation and concentrated disadvantage (Sampson and Loeffler, 2010).

The broader social and political conditions that were specific to the 1960s and 1970s when the penal apparatus commenced its expansionary trajectory are characterised by a number of major overlapping developments (see National Research Council, 2014: chapter 4). Chief amongst them were the mutually entangled political responses to the Civil Rights Movement, ethnoracial division, and the ideological trope of "law and order". The degree to which these motivated the harsh turn in penal policy was subtended by rising public anxiety surrounding upward-trending crime rates coupled with a consistent bipartisan commitment to be "tough on crime" (Beckett, 1997; Tonry, 1999; Smith, 2004). The tightened institutionalisation of corporate power, the socioeconomic distress associated with deindustrialisation, and accompanying spillover effects all fed into the punitive orientation of social policy.

This is conveyed by the unfolding of the three (in)famous "wars" that permeated the mainstream policy discourse of the 1960s and the 1970s, namely the "war on poverty", the "war on crime", and the "war on drugs". Upon their initial launch, President Johnson explicitly linked them together and stressed the need to address the "root causes" of crime through investment not only in law enforcement but also in education, health, and broader welfare issues. And yet, in a period of immense political turbulence, the war on poverty readily morphed into a war on the poor as such. The interac-

tion between ethno-racial division and electoral politics saw the Civil Rights Movements divide the Democratic Party at a time when Republicans successfully effected a powerful political and electoral realignment surrounding the nexus of racial anxiety and class division. Most notably, the purported equivalence between the political mobilisation of African Americans on the one hand and the "problem of street crime" on the other was an ideological notion that also penetrated Democratic circles, to the point that any collective commitment to addressing the "root causes" of violence was replaced with the language of "law and order", espousing a punitive treatment of racialised poverty.

The articulation, implementation, and enforcement of criminal justice policy thus went from being more or less exclusively a matter for state and local authorities to becoming a major preoccupation of the federal government. This remarkable shift from protective to punitive interventionism was legitimised through artificially inflated crime statistics – as local police agencies were incentivised to report more crime in return for federally sponsored infusions of money and equipment (Thompson, 2010: 727) – serving as fodder for heightened public anxiety surrounding crime and attendant "social problems". This simultaneously undermined the use of social and economic programmes to reduce crime and poverty, as President Johnson's "Great Society" programmes were increasingly portrayed not only as unsuccessful but as fundamentally flawed in their design and intent. In the process, the Republican Party, in pursuing its so-called "southern strategy", successfully deployed a persistent motif in the nation's cultural imaginary that associated "the problem of crime" with (poor) Blacks, who in turn were linked to the

"problem of welfare dependency". The only ethically meaningful and politically potent solution to these interlinked "problems" was, according to this line of argument, the introduction of harsh punishments for any disturbance to "public order".

The punitive political reorientation seen in both "wars" on poverty and crime was further distilled in the "war on drugs". The latter was only in part a response to exogenous shocks to local drug environments[5] and served to escalate the criminalisation of racialised poverty in ways that transcended party lines. Perhaps the most pronounced illustration of this was seen in President Clinton's ratification in 1994 of a 100-to-1 sentencing disparity between crack and powder cocaine – two virtually identical drugs that differed only in that the former was consumed almost exclusively by African Americans in areas of concentrated poverty, whereas the latter was viewed as an innocuous ingredient of White (upper) middle-class leisure time (Frank, 2016).

2.4 The anatomy of American social policy

Penal expansion in the United States has been a central part of a broader social policy repertoire with three principal components. First, the social safety net for society's most vulnerable is characterised by a consistent political emphasis on workfare over welfare, entailing the deployment of public assistance as a means of work enforcement (Peck, 2001). The most inci-

[5] The major exogenous shock to drug availability came not "from below" but "from above" when, in late 1995, opioids like OxyContin were not only deregulated by the U.S. Food and Drug Administration but aggressively advertised and promoted through media and physician marketing channels (see e.g. Hadland et al., 2019).

sive sociological articulation of this phenomenon is furnished by Frances Fox Piven and Richard Cloward, who construe periodically expanding and contracting relief rolls as serving the twofold purpose of muting civil disorder and enforcing low-wage work norms (Piven and Cloward, 1993). The authors note how relief arrangements are augmented during times of social and political turbulence, wrought by the endogenous dislocations of capitalism, only to be curtailed during times of stability and continuity, and they chronicle the countercyclical evolution of public assistance in the United States, from rapid expansions under the Great Depression in the 1930s and the turmoil of the 1960s Civil Rights era, to austere contractions during periods of relative stability in the 1940s and 1950s, and then again in the 1990s.

The latest historical inflection of this welfare trajectory has been subject to extensive scrutiny and debate. Median monthly Aid to Families with Dependent Children (AFDC) welfare payments to poor single mothers went down from $221 in 1970 to $119 in 1995, measured in constant dollars, which is equivalent to a net decline of over 50% in purchasing power. Regional variation in this decline ranged from 48% in Michigan to 68% in Texas. Maximum cash payment in the median state in 1994 equalled 38% of the federal poverty line, and only 69% when combined with food stamps and other welfare arrangements, thus designed to disincentivise "welfare dependency" (Wacquant, 2009: 49–54). In 1996, coverage of poor American households eligible to receive benefits slipped below the 50% threshold, at which point the AFDC itself was repealed and replaced by Temporary Assistance for Needy Families (TANF) under the Personal Responsibility and Work Opportunity Act (PRWOA) of President Clinton's Welfare Reform,

which ended 61 years of poor families' entitlement to federal welfare benefits by downsizing social transfers and multiplying the bureaucratic obstacles to meeting relevant eligibility criteria (Hays, 2003). Between 1970 and 1996, the value of cash assistance benefits for poor families with children had already fallen by more than 40% in real terms in the vast majority of U.S. states. Since the introduction of TANF, they have tumbled by at least another 20%, to the point where, as of July 2016, TANF benefits for a family of three with no other cash income were below half of the official poverty line in every state, and most states' benefits were below 30% of the poverty line (Stanley et al., 2016).

The PRWOA abolished lone mothers' right to public assistance as stipulated by the 1935 Social Security Act, curtailed support to a lifetime cap of five years, and imposed the obligation for recipients of aid to work within two years (at most). It devolved the administrative burden of assistance to individual states of the Union and their counties, whilst incentivising policies designed to reduce the number of recipients through the imposition of restrictive eligibility criteria and reduced support. Moreover, it instituted block grants that pegged cash endowments to fixed annual amounts, preventing welfare from operating in a countercyclical fashion in times of recession or crisis. Finally, it legally excludes "fringe" populations – most notably former drug convicts, but also foreign residents, poor disabled children, or unwed teen mothers who refuse to live with their parents (Wacquant, 2009: 91; Danziger, 2010). Public aid offices, under the aegis of health professionals without any medical training, were instructed upon its implementation to set medical boundaries to the effect that thousands of disabled individ-

uals were deemed "fit for work" and hence denied social assistance. This was buttressed and amplified by long-winded, tedious, and disparaging administrative procedures designed to discourage applications and disillusion potential recipients, a strategy proven successful by that fact that, by 2014, only 23 families received TANF benefits for every 100 poor families with children, as compared to 68 for every 100 poor families in 1996 (Stanley et al., 2016).[6]

Most of the social scientific literature on recent American welfare state policy focuses on the transition from AFDC to TANF, without considering the wider gamut of federal social policy programmes that notably include the Earned Income Tax Credit (EITC) and the Supplemental Nutrition Assistance Program (SNAP). A sole focus on TANF is likely to divert attention from the fact that the 1996 Welfare Reform epitomised not so much the dismantling but the reconfiguration of the social wing of the state anchored in a shift from a need-based to a work-based policy paradigm (Tach and Edin, 2017; see also Moffitt, 2015). The entrenchment of workfare is conveyed by how, according to the Organisation for Economic Co-operation and Development, the average net replacement rate for low-wage workers receiving

[6] The bureaucratic pressure exerted on American welfare recipients is conveyed by Susan Hays' ethnography of poor single mothers: "The first thing you see on entering the Arbordale welfare office is a large red banner, 12 feet long, 2 feet high, reading, 'HOW MANY MONTHS DO YOU HAVE LEFT?' Underneath that banner is a listing of jobs available in the area – receptionist, night clerk, fast food server, cashier, waitress, data entry personnel, beautician, forklift operator. In most cases, the hours, benefits, and pay rates are not listed. The message is unmistakable: you must find a job, find it soon (before your months run out), and accept whatever wages or hours you can get" (Hays, 2003: 32).

unemployment insurance in the United States is 67% for a maximum duration of six months or, in the case of nine states, only up to three months (Burtless, 2018). Typically, unemployment insurance is disbursed for 15 weeks at a stagnating real average value of around $300 (in current dollars), whilst coverage has shrunk from over 75% of wage earners following the 1935 Social Security Act to less than 40% in 1995. At the same time, the termination of the Comprehensive Education and Training Act, designed to reintegrate the unemployed, eliminated almost half a million jobs for unskilled wage earners in 1984, whilst overall federal spending on job training also dwindled after 1975 (Wacquant, 2009: 52–53). However, as evidenced by Figure 2.1, despite the fact that classic welfare support has tumbled, the reach of federal government programmes has expanded in tandem with increased spending on means-tested assistance measures (Congressional Budget Office, 2013), which have largely targeted workers living just above the poverty line. The consequence has been the emergence of a "middle-class" safety net (see Reeves and Pulliam, 2018) that rests on the use of public policy as a vector of discipline, deploying mandatory work assignments as a powerful institutional mechanism by which precarious wage labour is generalised in the nether regions of social space (Corcoran et al., 2000; Danziger et al., 2000; Collins and Mayer, 2010; see also Keith-Jennings and Chaudry, 2018; Krueger and Posner, 2018; The Economist, 2018).

The second component of this policy repertoire is the punitive treatment of those who fall between the cracks of the safety net. This punitive treatment is largely gendered, with the male side of the emergent "precariat" being enmeshed in the criminal justice system and the female side being subjected

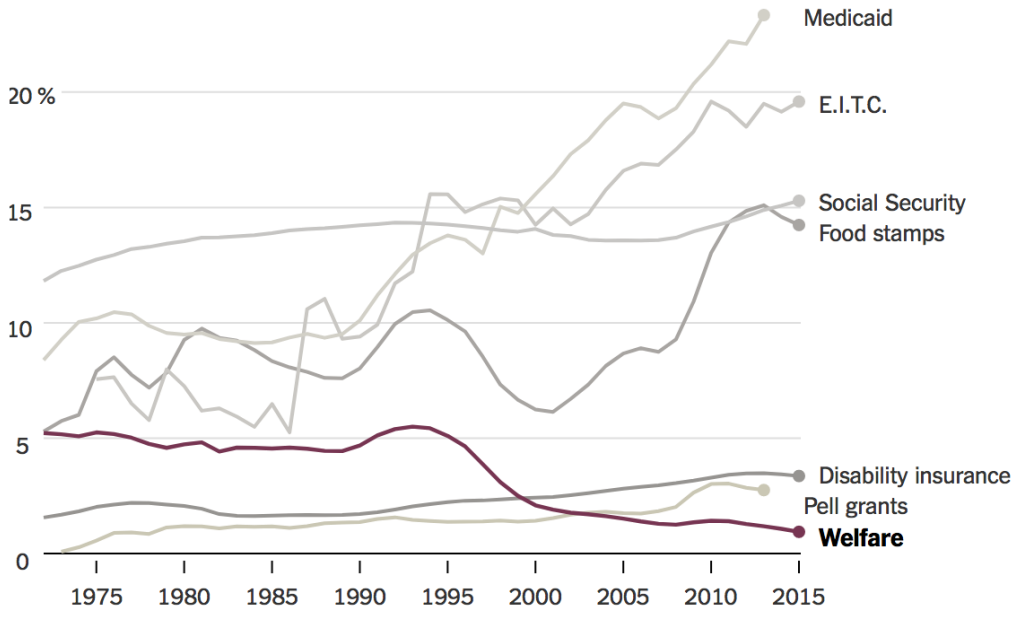

Figure 2.1: *Share of Americans using government programmes. Source: replication of Mettler (2018a) in The New York Times (Badger, 2018).*

not only to persistent workfarism but also to spatial reshuffling by means of aggressive gentrification. Coupled with stagnating incomes and rising housing costs, eviction constitutes a consequential form of social punishment for deprived female-headed households. It leads to the inability to secure decent housing for their families, further downward mobility, and heightened risk of homelessness (Desmond, 2012; 2016). However, the retrenchment of the protective wing of the state is accompanied by the expansion of its punitive wing (cf. Beckett and Western, 2001) not only for a fractured working class but also for other "marginal" populations. Most notably, successive waves of "deinstitutionalisation" during the 1960s and 1970s have led to the mentally ill having found shelter in jails after being thrown on the streets by downsized hospitals and psychiatric institutions. The number of psychiatric hospital patients nosedived by close to 90% between the mid-1950s and the mid-1990s (from over half a million to less than 70,000), yet those same individuals seem to have reappeared in the country's jails and prisons, typically arraigned for public order infractions that reflect their mental impairment and psychological suffering (Wacquant, 2009: 90). The estimated prevalence of mental illness exceeds 60% in county jails and 50% in state prisons, and is especially high amongst White inmates, whilst the prevalence of serious chronic illnesses, such as major affective disorders or schizophrenia, is estimated to be roughly one-fifth of the total carceral population (National Research Council, 2014: 204–205, Table 7-1).

The third component of the policy repertoire is the making of a generous welfare system for the wealthy. As indicated by Figure 2.2, the American welfare state has a dual structure. Insofar as social policy may be defined

as "any government effort to deliver economic security to citizens through the protection against income loss and the guarantee of a minimum standard of living" (Faricy, 2016: 3), tax expenditures, a form of off-budget spending executed through the tax code, emerge as a major mechanism whereby the American state consistently provides support for society's most privileged (see also Massey, 2008: 180–185). In 2004 alone, $64 billion were made in fiscal deductions for mortgage interest payments and real estate taxes (as compared to $17 billion going to welfare payments to the poor, $25 billion to food stamps, and $7.5 billion to child nutrition assistance), up to half of which went to the top income ventile (Wacquant, 2009: 42). Although the notion of "welfare" in the United States is most closely associated with Reagan's (in)famous invocation of a woman from Chicago's Black Belt who "has eighty names, thirty addresses, twelve Social Security cards and is collecting veterans' benefits on four nonexisting deceased husbands" (cited in Faricy, 2016: 11; see also Gilens, 1999), the private social system in the United States is a generous welfare arrangement backed by the federal state, principally accruing substantial benefits to wealthy and typically White members of the corporate community (Faricy, 2016: 11). This is evidenced by how, in 2015, close to 70% of all tax expenditures on pension benefits went to the top income quintile, compared to only 0.6% to the bottom quintile (*ibid*: 13). Tax expenditures are inscribed in the tax code for health care, education, and old-age pensions (*ibid*: 108–113), with pension benefits alone equating to $86 billion for the wealthiest, i.e. threefold the amount of the bottom 80% of the income distribution. As a share of total tax expenditures, over half (51%) go to the highest income quintile, compared to 8% to the bottom

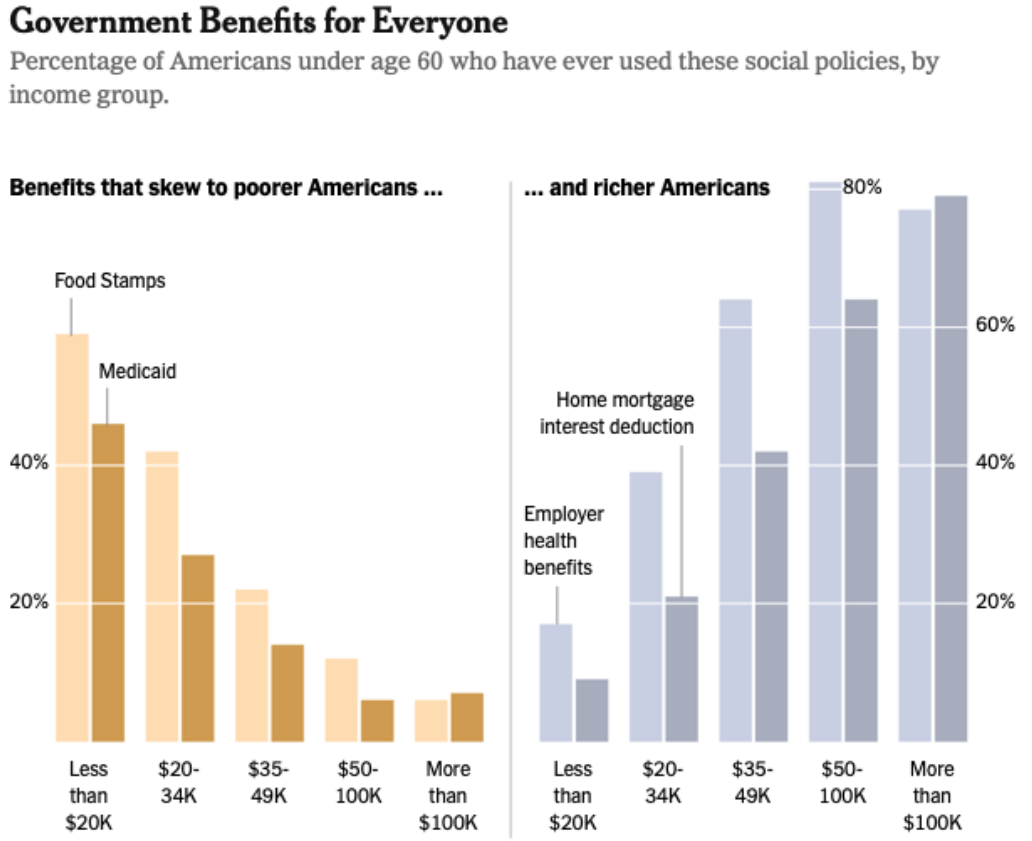

Figure 2.2: *The distribution of government benefits across the American income distribution. Source: Mettler (2018b).*

quintile (*ibid*: 13). Tax expenditures contribute significantly to undoing tax progressivity in that they reduce tax rates more substantially for individuals and households who pay higher marginal rates. In short, the bicephalous welfare system effectively serves to redistribute resources to the top of the social order.

When viewed as a whole, the anatomy of social policy is a likely driver of deepening inequality in the United States through a distinct form of in-

stitutional imbrication that harnesses both "hands" of the state (Bourdieu, 2002) to practice disciplinary paternalism at the bottom of the class structure and protective maternalism at the top.[7] Anchored in the politics of federalism, there is regional variation in the kind of institutional imbrication that is operant in a given city, county, or state. Such variation is historically informed by deeply entrenched organisational cultures that shape the political use of public and private institutions that, in turn, influence population health outcomes (Kunitz, 2015). Such structural variation can help account for the geographically patterned mortality burden across the United States, as visualised in the Introduction (see Figures 1, 3, and 4), potentially with even greater predictive power than a sole focus on economic deprivation or decline would allow. This is conveyed by Stephen Kunitz' comparative historical analysis of two pairs of states with similar levels of per capita and median household incomes but with contrasting institutional arrangements,

[7] Therborn (2013) distinguishes between four mechanisms by which inequalities are generated. *Distanciation* is construed as a systemic process whereby social distances between "winners" and "losers" are spawned, fuelled by institutionalised structures of reward and retribution, promotion and penalty, privilege and punishment. *Exclusion* refers to the process by which selected social agents are hindered from fully partaking in social life through obstructed access to symbolic and material goods, typically via discriminatory expulsion or social "caging". *Hierarchisation* designates the formal organisation of socially defined relations of superiority and inferiority, manifest in intra-organisational rankings and distinctions. And finally, *exploitation* entails a distinctly asymmetrical relation of domination between two parties whereby one, through authority or coercion, extracts (surplus) value from the other (*ibid*: 62, Table 4). Based on the above, all four – but especially the first two – of these mechanisms appear to be deeply inscribed in the very fabric of American social policy.

namely North and South Dakota on the one hand, and Kentucky and West Virginia on the other. Nested within the Border states, there is significantly greater resource inequality (as measured by income and education), existential inequality (as measured by the status of Native Americans, to which the enduring legacy of slavery can be added), and vital inequality (as measured by levels of mortality) than in the Dakotas. Moreover, typical socioeconomic variables are strongly associated with aggregate mortality trends in Kentucky and West Virginia, but not so on the Northern Plains. What seems to account for these divergent patterns is, at least in part, "the propensity to use both governmental and non-governmental institutions to address social conditions [and community problems]" (*ibid*: 54; see pages 124–146), notably in the form of mobilisation around civic conceptions of the public good.

It just so happens that both Border states have experienced sharp increases in incarceration rates that continue today, even when the national average has started to stagnate and decline under the press of overcrowded facilities and tightening state budgets. In Kentucky, the prison admissions rate has gone from around 100 per 100,000 working-age residents in 1980 to just under 700 in 2016, whilst the jail incarceration rate has grown sixfold from around 50 to approximately 300 per 100,000 (Kang-Brown et al., 2018: 31, Figure 9). In West Virginia, the overall prison incarceration rate has roughly quintupled from around 70 to just under 400 inmates per 100,000 population between 1978 and 2014, and the jail incarceration rate has nearly tripled to reach around 180 per 100,000 (Prison Policy Initiative, n.d.). Total incarceration rates in the two states are at an astounding 869 and 690 inmates per 100,000 population, respectively. At the same time, nine of the

top ten counties nationwide with the highest level and eight of the top ten counties with the largest increase since 1980 in age-standardised mortality rates from drug use disorders are located in either Kentucky or West Virginia (Dwyer-Lindgren et al., 2018). Conversely, six of the top ten counties with the lowest drug-related mortality rates are located in the Dakotas, where incarceration rates are far below the national average in North Dakota (at 560 per 100,000 population), although above average in South Dakota (855 per 100,000). However, five of the top ten counties with the smallest increase in such deaths are located in New York, where the urban prison admissions rate has dropped by 50% since the mid-1990s (Kang-Brown et al., 2018: 22, Figure 5), placing the overall New York incarceration rate well below the national average (at 443 per 100,000 population). On this basis, as I will look to empirically substantiate in Part II below, it is unlikely by chance that Kentucky and West Virginia have borne the brunt of the ongoing overdose epidemic, which has aggravated the already deep-seated mortality disadvantage carried by their populations over many decades.

2.5 Conclusion

I have adumbrated a number of social, political, and economic forces that generate unequal life chances. The hypothesised mechanisms by which these produce vital inequality will be outlined in the next chapter. I have highlighted that capitalism produces endogenous dislocations that, in turn, act upon some of the major social determinants of health and illness. Most notably, in the wake of rapid industrial restructuring, the social wing of

the state stems the tide of economic dislocation by deploying workfare as a means of effecting the transition from manufacturing to service sector employment. Thus an emergent subproletariat is springboarded into precarious wage work, whilst for those who fall through the cracks, punitive measures are rolled out. The disciplinary aspect of this policy repertoire has been a major force behind an increasingly dualised social structure, as illustrated by the sharp rise in the number of households whose incomes are located below the World Bank's $2-a-day poverty line (Edin and Shaefer, 2015). Indeed, the number of children in such poverty has more than doubled since the 1990s as poor single mothers – the female counterpart to those who typically populate the country's jails and prisons – have experienced plummeting real incomes (Tach and Edin, 2017: 544–545) and chronic economic insecurity (Collins and Mayer, 2010). I have emphasised the important but oft-neglected role of corporate power, anchored in and buttressed by state policy, in driving this deepening gulf between the top and the bottom of the socioeconomic order.

Chapter 3

Pathways and mechanisms

3.1 Introduction

One of the main points that emerged from the previous chapter was that one of the principal determinants of inequality appears to be not only economic decline but *political responses to economic decline*. The literature on the health effects of resource shocks associated with deindustrialisation (Hopper et al., 1985; Hamilton et al., 1990; Wagner, 1991; Byrne, 1995; Aghion et al., 2016), trade liberalisation (Pierce and Schott, 2016b; Barlow et al., 2017; McNamara, 2017), or broader labour market conditions is vast (see Avendano and Berkman [2014] for an overview). However, there are various possible welfare state responses to such resource shocks (e.g. Iversen and Cusack, 2000) that can moderate or magnify deleterious impacts (Stuckler and Basu, 2013). A distinctive feature of American social policy is its punitive mode of poverty regulation in the wake of economic decline, epitomised by high rates of incarceration. In this chapter and the ones to follow, I will therefore

construe the operations of the penal state as a window through which the broader ramifications of punitive social policy can be grasped (see Nosrati and Marmot, 2019). By detailing its corrosive impact on the most vulnerable nether regions of social space, and thus on the "fundamental causes" of ill health (Link and Phelan, 1995), I will briefly delineate the hypothesised pathways and mechanisms through which penal expansion can shape vital inequality, and how it contributes to the making of what Case and Deaton (2017) call "cumulative disadvantage".

3.2 Poverty, punishment, and poor health

The dynamics of (un)employment, social marginality, and incarceration are mutually entangled (see e.g. Western et al., 2006; Western and Muller, 2013). Amongst incarcerated men between the ages of 27 and 42, 57% earn less than $22,500 per annum prior to incarceration. The median incarcerated man has a pre-incarceration income that is less than half of that of the median non-incarcerated man (Rabuy and Kopf, 2015). For African American ex-prisoners, employment duration decreases from 35 weeks per year to 21, hourly wages sink from $10.25 to $9.25, and consequently annual earnings tumble by over 45% from an average of $13,000 before to $7,000 after imprisonment. Controlling for drug use, but also age, education, work experience, industry, region of the country, public sector employment, union status, marital status, school enrolment, urban residence, local unemployment, year, and education-by-year interactions, incarceration reduces annual employment by 9.7% for European Americans, by 13.7% for Hispanics, and

by 15.1% for African Americans. For hourly wages, the corresponding figures equal 16.3%, 25.7%, and 12.4%, whilst annual income is reduced by 35.9%, 32.2%, and 36.9%, respectively. Such results are also seen in employment prospects, in terms of wage growth and tenure, leading to substantial negative aggregate effects on lifetime earnings (Western, 2006: 116, 119, 124–127). Former convicts experience overall unemployment rates of 27%, which exceeds the total unemployment peak of the Great Recession in 1933 (Couloute and Kopf, 2018). Once released from prison, former inmates of colour, notwithstanding their prior disadvantage in the labour market, see their job application success rate drop by 65% (Pager, 2003: 958) and often slip out of the official labour force, which further contributes to hidden joblessness that evades official statistics (Western, 2006: 87).

Whereas rates of homelessness are 21 per 10,000 population for the general public, for those who have been incarcerated once, it is 141 per 10,000, and for those with multiple encounters with the criminal justice system, the number is 279. In other words, for those who have been incarcerated more than once, homelessness rates are 13 times that of the general public. Moreover, according to data from the Bureau of Justice Statistics, high rates of recidivism, fostered by the active criminalisation of poverty (Thompson, 2010), imply that almost half of those released from prison are rearrested within 1 year, over two-thirds within 3 years, and over 80% within 9 years, thus creating a vicious cycle of punishment and poverty (Couloute, 2018).

Previous research has not only shown that former inmates experience mortality rates close to thirteenfold that of the comparable populace – and are especially vulnerable during the first two weeks post-release (Binswanger

et al., 2007; Zlodre and Fazel, 2012) – but also that high incarceration rates exert cascading effects across generations, local communities, and other networks of current or former incarcerated persons (for recent reviews, see National Research Council, 2014: chapters 7–10; Massoglia and Pridemore, 2015; Wildeman and Wang, 2017). Put differently, the experience of incarceration may be traumatic in and of itself, both for those who are incarcerated and for their families, friends, and broader social connections (see Western, 2018). The incarceration of a family member has been shown to impair the wellbeing of non-incarcerated partners and children (Pettit and Western, 2004; Freudenberg et al., 2005; Wildeman, 2009; Wildeman and Muller, 2012; Turney, 2014), notably due to declining household income, reduced parental investment, unstable social relationships, and psychosocial stress (see also Arditti et al., 2003; Christian, 2005; Lopoo and Western, 2005; Comfort, 2007). Net of purely material factors, a mother's risk of a major depressive episode and her level of life dissatisfaction is heightened as a result of her partner's incarceration (Wildeman et al., 2012). Whilst marriage is relatively uncommon in the carceral population (14% of European Americans, 22% of Hispanics, and 11% of African Americans in prison are married, respectively), fatherhood is not (figures for fatherhood equal 64%, 81%, and 70%, respectively). Thus between 1980 and 2000, the total number of children with a father in prison increased sixfold to reach over 2 million at the turn of the century, i.e. almost 3% of all children nationwide. Amongst these, the cumulative risk of experiencing parental incarceration by age 14 amongst African American children born to high-school dropouts exceeds 50% (Wildeman, 2009).

At the community level, the criminal justice system plays a pivotal role in shaping the trajectories of neighbourhoods by removing prime age men from their local communities, tearing families apart, and disrupting social networks (Western, 2006; Clear, 2007; Wacquant, 2009; Sampson and Loeffler, 2010; National Research Council, 2014; Western, 2018). When coupled with socioeconomic relegation, the operations of the penal state constitute an upstream determinant of "despair" whereby regular exposures to neighbourhood violence, unstable social and family relationships, and psychosocial stress trigger destructive (health) behaviours (Clear, 2008; Merall et al., 2010; Thompson, 2010; Drakulich et al., 2012; Turney, 2014). Symbolically, the existence of former prisoners, their friends, and their family is stained by the stigma that incarceration stamps on both their past and their future. In crushing combination with deteriorating employment parameters and deepened material hardship, this consolidates what Goffman (1968) dubs a "spoiled identity" – which in itself is a social determinant of health (Phelan et al., 2014).

Indeed, one of the simplest yet most consequential effects of incarceration is the increasing invisibility of the poor from the public sphere (Patillo et al., 2004; Western 2006; National Research Council, 2014). Physically, the victims of punitive social policy are confined to the spatial container that is a prison or to the perimeter of highly segregated neighbourhoods (cf. Massey and Denton, 1994). Socioeconomically, their lives are corralled in the bottom layers of social space. Carceral populations are entirely omitted from national surveys deployed to track economic trends (Western 2006: 87), thus shrouding a persistent form of exclusionary closure. For young African

American men at century's dawn, unemployment jumps from one-fourth to one-third once incarceration is taken into account (*ibid*: 90). For prime age men of colour without a high school diploma, the same figure swells from 41% to 65%, meaning two out of three Black male dropouts are absent from the labour market, whereof nearly half is due to incarceration (*ibid*: 91). Current and former convicts become politically disenfranchised, meaning they are not only "locked up" but also "locked out" (Manza and Uggen, 2008), with potentially profound consequences for vital inequality (see Beckfield, 2018: 35–39, 84–88). They typically lose eligibility for the meagre social assistance available to the most vulnerable and the stigma they carry percolates to the very core of their existence, which is symbolically abrogated and annulled.

Just like the gradual decomposition of the American working class under the press of deindustrialisation has evoked metaphors of collective defeat and subjugation (see Milkman, 1997; Cowie and Heathcott, 2003; High and Lewis, 2007; Doussard et al., 2009), so do these interlocking social processes, when viewed in tandem, ferment the sense of subjective demoralisation that permeates the bottom rungs of the socioeconomic landscape. They create and compound diverse modalities of "social sundering" (Therborn, 2013: 22–28), dissolve the prospect of any collective cohesion or mobilisation, and precipitate a form of social death – one that, as we shall see in the forthcoming chapters, is accompanied by physical deaths. In short, (hyper)incarceration means more than just penal confinement. It is a powerful institutional force that mediates and modifies, amplifies and aggravates the nexus of resource inequality, existential inequality, and vital inequality. Based on existing evi-

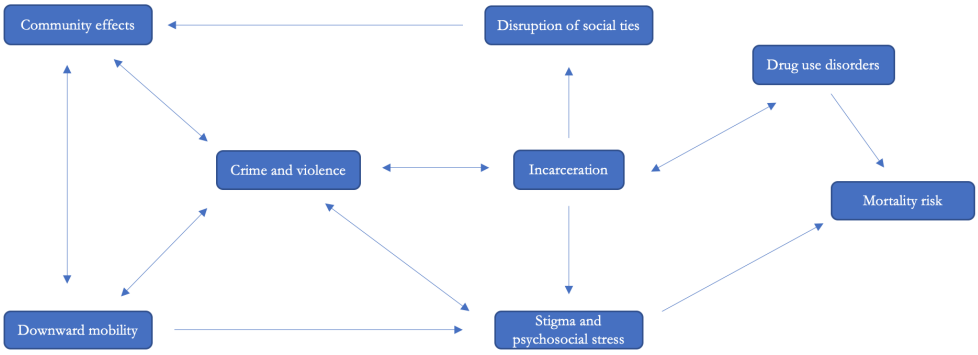

Figure 3.1: *Hypothesised pathway(s) by which high incarceration rates are associated with mortality risk: both jail and prison incarceration can lead to both network disruption and stigma, with cascading community effects; these include crime and violence, as well as both individual and collective downward mobility; these factors coalesce into a self-reinforcing system conducive to psychosocial stress (or "despair").*

dence on how punitive social policy can lead to vital inequality, a proposed set of pathways is visualised in Figure 3.1. These pathways are hypothesised to be operant over and above the endogenous health profiles of the incarcerated (see National Research Council, 2014: 204–213). As I will discuss in Part II, and in light of the spillover effects described above, punitive social policy likely has the power to shape aggregate patterns of vital inequality beyond its impact on those individuals who pass through the criminal justice system.

3.3 Embodied inequality

One of the critiques directed at the analytic framework offered by the political economy of public health is the question of its biological plausibility (Krieger, 2011: 167–191, 213–234). By virtue of primarily lending its focus to distal causes of health and illness that seem far removed from the lived experience of individual organisms, the framework has been described as "biologically opaque, affording little insight into the biophysical phenomena relevant to translating societal conditions into population patterns of health, disease and well-being" (*ibid*: 213). Thus far, I have briefly described the cascading causal chains whereby upstream political and economic factors influence health through a number of meso-level mediators, especially group-level mechanisms of psychosocial distress that trigger deleterious health behaviours or in themselves heighten mortality risk. There is, however, an emerging field of human social genomics (see Cole, 2009; Slavich and Cole, 2013; Cole, 2014) that may elucidate how both material and symbolic components of our social habitat shape human gene expression in ways that profoundly affect a range of complex phenotypes and disease susceptibility (for a critical discussion, see Shostak and Beckfield, 2015). Although not pursued further in the coming chapters, I offer a brief sketch of these scientific developments that offer promising avenues for future research on embodied inequality (cf. Wolfe et al., 2012).

Gene expression is regulated by intracellular proteins (transcription factors) that activate genes in the form of RNA in response to a wide variety of endogenous and exogenous signals. Extracellular signals, such as stress hormones (e.g. adrenaline or cortisol) secreted into the bloodstream in response

to social adversity (say, sudden job loss or the imprisonment of a family member), can elicit changes in genome-wide transcriptional dynamics by up-regulating entire gene profiles whilst down-regulating others. Genes that are particularly sensitive to social regulation have been observed in the immune system, where a skewing of the basal transcriptome in leukocytes (white blood cells) has been linked to enhanced production of pro-inflammatory cytokines coupled with silenced anti-viral immune responses in contexts of social isolation (e.g. Cole et al., 2007), acute and chronic stress (e.g. Powell et al., 2013; Murphy et al., 2015), childhood adversity (e.g. Miller et al., 2009), socioeconomic disadvantage (e.g. Chen et al., 2009), or low relative status (e.g. Tung et al., 2012; Snyder-Mackler et al., 2016) – all of which are at work in the intertwined social processes described above. Up-regulated pro-inflammatory immune response genes increase susceptibility to conditions such as cardiovascular disease and cancer, whilst down-regulated anti-viral immune response genes render humans vulnerable to viral infections.

Combined with prolonged exposure to (socially and spatially) concentrated hardship, this so-called "conserved transcriptional response to adversity" amplifies the cumulative biological burden known as allostatic (over)load (see Seeman et al., 2001) whereby disruptions in the lived experience of social agents are deposited in the human body in the form of neuroendocrine traits that govern core pathogenic parameters. Amongst the manifold molecular pathways involved in such "social signal transduction" (see Slavich and Cole, 2013; Slavich and Irwin 2014), epigenetic processes, notably chemical modifications of histone proteins, have been shown to influence the regulation of gene expression in ways that further reflect the transcriptional embedding

of social experiences (e.g. Champagne, 2010; Meaney and Ferguson-Smith, 2010). For instance, socially differentiated patterns of DNA methylation (the addition of a methyl group to DNA) within the pathways described above have been shown to contribute to the up- or down-regulation of immune response genes (e.g. Liu et al., 2008).

These novel insights, located at the interface of the social and biological sciences, form a nascent field of enquiry filled with promises (Kubzansky et al., 2014; Moore, 2015) but also with deep uncertainties (Miller, 2010). However, they suggest emergent scientific opportunities to empirically expand and conceptually unify the study of embodied inequality in a way that entails the joint inclusion of the social and the biological, the symbolic and the material in our analytic purview. They subtly point to what Claude Lévi-Strauss, in his anthropology of symbolic efficacy, famously describes as "a certain 'inductive property', by which formally homologous structures, built out of different materials at different levels of life – organic processes, unconscious mind, rational thought – are related to one another" (Lévi-Strauss, 1974: 201). This very "inductive property", onto which social genomics is breathing fresh understanding, is what enables embodied social life through "a biological [...] reading of social properties and a social reading of [biological] properties, thus leading to a social re-use of biological properties and a biological re-use of social properties" (Bourdieu, 1992: 79).

3.4 Conclusion

In this chapter, I have reviewed existing evidence on the role of incarceration in shaping the interface between resource inequality, existential inequality, and vital inequality. Punitive political responses to economic decline act on the social determinants of health, chief amongst them unemployment, poverty, and stigma, and generate vicious spirals of relegation and despair. Moreover, in light of recent scholarship, they constitute a locus of organic tension, a veritable "socio-biological phenomenon", in the sense of Marcel Mauss (2013). Politics and policies must therefore be conceptualised as part of the epigenome, i.e. as sites of lived experience that actively shape human gene expression and organic functioning (Beckfield, 2018: xxix).

Part II

Analysis

Chapter 4

Jails, prisons, and the American overdose epidemic

4.1 Introduction

America is in the grip of an overdose epidemic, with age-standardised mortality rates from drug use disorders growing more than 600% since 1980 and currently being the second leading cause of death nationwide for those between the ages of 15 and 49. Overdose deaths have increased in every single county, but at vastly different rates ranging from 8% to over 8000% (Dwyer-Lindgren et al., 2018). In this chapter, I empirically test the hypothesis according to which incarceration – in the form of jail and prison admissions rates – can help explain the rise of such mortality rates and the spatial variation in their prevalence, net of other confounding factors such as median household income, crime, or local opioid prescription rates. Using hitherto unavailable panel data, I examine these associations for the first time, looking

both at variation within and between counties, finding that jail and prison incarceration rates are major predictors of drug-related mortality.

4.2 Empirical strategy

Data

My outcome variable is the annual age-standardised mortality rate from drug use disorders per 100,000 population for 2,640 U.S. counties between 1980 and 2014. These recently released public-use data from the Institute for Health Metrics and Evaluation are generated from death registration data collected by the National Vital Statistics System using the Global Burden of Diseases, Injuries, and Risk Factors Study cause list. This cause list is composed of hundreds of mutually exclusive causes of death that are matched onto the International Classification of Diseases, ninth and tenth revisions (ICD-9, ICD-10; for details, see eTable3 in the Supplement of Dwyer-Lindgren et al., 2018). The novelty of this data set is that it uses machine learning algorithms and small-area estimation methods to rectify potentially misleading spatial and temporal patterns in mortality trends across geographical units due to poor-quality death codes in previous data collections. It also offers age-standardised mortality estimates by cause of death for all U.S. counties since 1980. This chapter focuses on age-standardised mortality rates from drug use disorders (ICD-9 codes 292–292.9, 304.0–304.83, 305, 305.1–305.93, 760.7–760.79, E850–E850.29; ICD-10 codes F11–F16.99, F18–F19.99, P04.4–

P04.49, P96.1, R78.1–R78.5).[1]

I use hitherto unavailable county-level jail and prison incarceration data from the Vera Institute of Justice (Kang-Brown, 2015; Subramanian et al., 2015; Hinds et al., 2018; Kang-Brown et al., 2018). Jail incarceration data, collected in the Census of Jails and the Annual Survey of Jails by the Bureau of Justice Statistics, are compiled to generate annual county-level rates per 100,000 residents aged 15–64 by the Vera Institute of Justice. Prison data from state corrections sources and the National Corrections Reporting Program by the Bureau of Justice Statistics are also compiled into annual county-level rates per 100,000 residents aged 15–64, although the earliest available data come from 1983 (Hinds et al., 2018). Prison data at the state level are collected in the annual National Prisoner Statistics survey by the Bureau of Justice Statistics. Six states – Alaska, Connecticut, Delaware, Hawaii, Rhode Island, and Vermont – are excluded from the analysis because they do not have local jail systems. In these states, the prison system operates the pre-trial detention and short incarceration sentences managed by county jails in other states. Thus, prison admissions data in those states are more comparable in magnitude to jail admissions data elsewhere, and the Bureau of Justice Statistics has not collected true prison admissions data in those states consistently. Due to certain discrepancies between different data sources in measuring county boundaries and accounting for changes to counties over time, the state of Virginia and a handful of counties from other

[1] It is worth noting that the composition of drug-related deaths has remained relatively stable over the study period, with the exception of mortality rates from prescription opioids, which have jumped up around the turn of the century to the point of overtaking heroin and cocaine as the leading cause of overdose deaths.

states are also excluded from the final analysis. Finally, salient outliers, potentially caused by measurement error in the Vera data set, especially in the early years of the panel (before 1983), are removed.

My control variables are drawn from the U.S. Census Bureau and the National Center for Health Statistics, and some of them are described in eTable2 of Dwyer-Lindgren and colleagues (2017). They include the following:

- Median county household income, adjusted for inflation.
- Fraction of the county population aged 25 or older who have completed high school.
- Fraction of the county population who are African American.
- Fraction of the county population who are Hispanics.
- Fraction of the county population who are of some other non-White ethnicity.
- Violent crime rate per 100,000 county population.
- For the years 2006-2014, I also use county-level retail opioid prescription rates dispensed per 100 persons as an additional control variable. These data are publicly available from the Centers for Disease Control and Prevention.

Of these control variables, the first five are based on various census estimates and are thus not available on an annual basis. To fill missing cells in the panel, linear interpolation is used to generate values between observations (Dwyer-Lindgren et al., 2017).

Descriptive statistics are presented in Table 4.1. The reader will note substantial inequalities in drug-related mortality, which on average is 4.9 deaths per 100,000 county residents, but with a standard deviation of 4.7. The lowest rate of drug deaths in any county-year is 0.3, whilst the highest rate lies at no less than 60.8 per 100,000 population. These inequalities, and their evolution over time, are visualised in Figure 4.1. Similarly, the jail and prison admissions rates are subject to substantial variation, ranging from around 20 to over 18,000 jail inmates per 100,000 and from 6 to over 730 prison inmates per 100,000 county residents. Comparable variation is also seen in median household income, the ethno-racial composition of counties, violent crime rates, and opioid prescription rates. A Spearman correlation matrix is displayed in Table 4.2, which shows relatively weak but expected correlations – for instance that median household income is negatively correlated with drug-related mortality and both jail and prison incarceration rates. However, these simple unadjusted correlations remain difficult to interpret and largely uninformative in the absence of a statistical approach that explicitly models how these variables relate to each other.

Methods

For all statistical models in this thesis, I adopt King's (1998) generalised model notation whereby

$$\begin{aligned} Y &\sim f(y \mid \theta, \alpha), \\ \theta &= g(X, \beta), \end{aligned} \tag{1}$$

Table 4.1: Descriptive statistics

Statistic	N	Mean	St. Dev.	Min	Max
Drug-related mortality rate	69,562	4.9	4.7	0.3	60.8
Jail admissions rate	67,172	7,018	3,822	20.2	18,445
Prison admissions rate	66,910	254.6	160.3	6.0	732.1
Median household income ($)	69,562	46,841	11,781	17,582	125,704
Fraction high school graduates	69,562	0.8	0.1	0.3	1.0
Fraction African Americans	69,562	0.1	0.2	0.0	0.9
Fraction Hispanics	69,562	0.1	0.1	0.0	1.0
Fraction other ethnicity	69,562	0.02	0.05	0.0	0.9
Violent crime rate	61,367	284.8	265.3	0.0	3,894
Opioid prescription rate	20,049	90.7	45.7	0.0	437.2

Notes: Rates are per 100,000 county residents; the mortality rate from drug use disorders is age-standardised; the county incarceration rates are per 100,000 population aged 15–64.

Table 4.2: Spearman correlation matrix

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Drug-related mortality rate	1									
2. Jail admissions rate	0.3	1								
3. Prison admissions rate	0.4	0.4	1							
4. Median household income	-0.05	-0.2	-0.2	1						
5. Fraction high school graduates	0.4	-0.1	-0.1	0.6	1					
6. Fraction African Americans	0.2	0.2	0.4	-0.2	-0.2	1				
7. Fraction Hispanics	0.4	0.2	0.3	0.2	0.3	0.2	1			
8. Fraction other ethnicity	0.3	0.1	0.1	0.3	0.5	0.02	0.6	1		
9. Violent crime rate	0.2	0.3	0.3	-0.01	-0.1	0.5	0.3	0.2	1	
10. Opioid prescription rate	0.5	0.3	0.3	-0.3	-0.2	0.2	-0.1	-0.1	0.2	1

where the first line designates the stochastic component and the second line designates the systematic component of the model for some random outcome variable Y (with realisation y), distributed according to some probability density defined by $f(\cdot)$. θ is the systematic feature of the density that varies with each observation, whilst α is usually a constant ancillary parameter. $g(\cdot)$ designates the functional form, and X and β are the covariates and effect parameters, respectively. This flexible notation allows for a wide variety of model specifications that are adaptable to different data sets and can be recalibrated to capture different quantities of interest. In this chapter, our primary quantity of interest will be the set of parameters that determine the expected value of the county-level age-standardised mortality rate from drug use disorders between 1980 and 2014. Using the above notation, this can be written as follows:

$$\begin{aligned} Y &\sim f_N(y_{it} \mid \mu_{it}, \sigma^2), \\ \mu_{it} &= \mathbb{E}(Y \mid X) = X\beta. \end{aligned} \tag{2}$$

Here the random outcome variable Y (with realisation y_{it}) is the mortality rate from drug use disorders, assumed to be distributed according the Normal probability density defined by f_N, conditional on a set of parameters; μ_{it} is the expected value ($\mathbb{E}$) of Y for county i at time t given a matrix of covariates, X, containing key predictors such as incarceration rates and economic decline, but also county and time fixed effects; β is the vector of effect parameters corresponding to each covariate; and σ^2 is the variance.

To examine the relationship between incarceration and deaths from drug use disorders, I deploy a number of different methodological strategies. First, I estimate a relatively conservative fixed effects panel regression. This model

identifies exclusively from within-county variation by "de-meaning" through entities over time. This has the virtue of controlling for any potential time-invariant confounders, even if these confounders are unobserved (or even unobservable). A major downside of this approach is that much, if not most, of the variation in mortality rates lies *between* rather than within counties, but this variation is entirely ignored by the fixed effects model. For this reason, but also to assess model dependence, I run a multilevel random effects "within-between" model (see Bell and Jones, 2015; de Leeuw and Meijer, 2008), which explicitly partitions the within- and between-county variance to reproduce the fixed effects estimates together with estimates from a "between" model. This is done by incorporating two versions of each covariate X into the model. The first, which will produce the equivalent of a fixed effects estimate, is the "de-meaned" version of X ($X_{it} - \bar{X}_i$). The second, which will lead to a "between" estimate, is simply the group (county) mean ($\bar{X}_i$). The multilevel approach also allows for the intercept term to vary across counties, and thus models rather than suppresses time-invariant county fixed effects.

Any statistical model will suffer from (at least) two sources of uncertainty (see King et al., 2000). First, there is *estimation uncertainty*, which stems from the lack of perfect knowledge of the parameter θ in Equation 1. This lack of knowledge derives from not having an infinite number of observations from which the exact, "true" value of θ can be calculated. Consequently, estimation uncertainty will be reduced as the sample size increases ($N \to \infty$). This form of uncertainty is typically acknowledged in empirical research in the form of standard errors or confidence intervals associated with a parameter estimate, $\hat{\theta}$, but is often neglected when computing additional quantities

of interest, such as predicted values, that are derived from such parameter estimates. A second form of uncertainty, known as *fundamental uncertainty*, is also rarely taken into account by researchers. Fundamental uncertainty designates intrinsic variability in the world – which exists regardless of our knowledge of θ – and is represented by the stochastic component in Equation 1. In other words, even if the researcher knows the exact data generating process, she will not be able to perfectly predict any and every future data point. This is simply due to the fact that the data generating process is subject to stochastic variability, albeit within the confines of some underlying probability distribution.

In order to better account for these forms of uncertainty, I follow two principal analytic steps. Before conducting any analysis, I deploy matching as a non-parametric form of pre-processing the data (Ho et al., 2007; Iacus et al., 2018). The goal of matching is to reduce inefficiency, bias, and model dependence, for instance in the form of uncertainty related to the functional form $g(\cdot)$ in Equation 1. It is a non-model-based approach to preparing the data for parametric analysis with a view to mimicking experimental research designs. In non-technical terms, matching seeks to select units of analysis (counties) that are similar if not identical to one another in all respects except for one: whether or not they are exposed to a key variable of interest. In the present case, the quantity of interest is the effect of high rates of incarceration on drug-related mortality, over and above the endogenous associations between incarceration and factors like income, education, or crime. Applying a matching algorithm will help "match" counties that share key characteristics, with the exception that some have high incarceration rates and others have

low incarceration rates. This will facilitate a more precise account of the link between penal expansion and drug-related deaths. In more technical terms, let Y_i designate the outcome variable of interest (drug-related mortality), let $T_i \in [0, 1]$ designate a dichotomous "treatment" variable (low versus high incarceration rates), and let X_i designate a series of pre-treatment covariates (income, education, crime, drug environment etc.). The "treatment effect" (TE) on a treated unit i is $TE_i = Y_i(T_i = 1) - Y_i(T_i = 0)$. However, the last term of this equation, $Y_i(T_i = 0)$, is an unobserved counterfactual. One can estimate this quantity with Y_j from control units (indexed by j) that are matched on relevant covariates (i.e. $X_i \approx X_j$) such that the estimated counterfactual quantity, $\hat{Y}_i(T_i = 0)$, is equal to $Y_j(T_j = 0)$. Unmatched units are pruned from the data set to improve empirical covariate balance between treatment and control groups in the sample, and the parametric model is applied to the pruned rather than to the raw data. As a result, the functional form of the parametric specification is subject to less arbitrary model dependence.

In the analysis below, I employ what is known as coarsened exact matching. This form of matching proceeds as follows. For lack of being able to match on *exact* values of continuous covariates, this algorithm temporarily "coarsens" the covariates X into sub-categories (e.g. quartiles). It then applies exact matching on the coarsened X, $c(X)$, before sorting observations into strata, each with unique values of $c(X)$. Any stratum with zero treated or control units is pruned from the data set. The algorithm then passes the original (uncoarsened) units – except for the pruned ones – on to the matched data set that is used in the parametric analysis. This particular approach to

matching possesses a number of desirable statistical properties that are not present in other matching methods and has been shown to produce robust results with good covariate balance (for details, see Iacus et al., 2012).

After extracting a pruned data set from the matching procedure, I adopt a simulation-based approach to presenting key quantities of interest, as proposed by Gary King and colleagues (King et al., 2000). Simulation can be used to obtain useful information about a chosen probability distribution by drawing random numbers from it. In looking to account for the two main types of uncertainty outlined above, it is possible to treat any model parameter estimates as features of their underlying sampling distribution. In fact, by the central limit theorem, the set of all model parameter estimates, represented by the stacked column vector $\hat{\gamma} = \{\hat{\beta}, \hat{\sigma^2}\}$, follows a multivariate normal distribution with mean equal to the model estimates and variance equal to the model variance-covariance matrix, $\hat{V}(\hat{\gamma})$. More formally, the distribution from which a simulated parameter value, $\tilde{\gamma}$, can be drawn is represented as follows:

$$\tilde{\gamma} \sim f_N(\hat{\gamma}, \hat{V}[\hat{\gamma}]). \tag{3}$$

To obtain expected values of Y (drug-related mortality) for counties with different values of the "treatment", T (incarceration), I do the following (for further details, see King et al., 2000):

1. I estimate a "between" model to further isolate between-county inequality, regressing Y on T. This is similar to the model displayed in Equation 2, except that observations are averaged over time for each county i (thus suppressing the time index) and that the dichotomous

treatment indicator T replaces the matrix of covariates X:[2]

$$
\begin{aligned}
Y &\sim f_N(y_i \mid \mu_i, \sigma^2), \\
\mu_i &= \mathbb{E}(Y \mid T) = T\beta.
\end{aligned} \tag{4}
$$

2. I simulate from the sampling distribution of the parameter estimates to incorporate estimation uncertainty by drawing M random numbers from the multivariate normal distribution, as specified in Equation 3.

3. For each simulated parameter value, I calculate the systematic component of the model in Equation 4, $\tilde{\mu} = T\tilde{\beta}$.

4. For each simulated systematic component, I draw another m separate random draws of the outcome variable, $\tilde{y}_k$ $(k = 1, \ldots, m)$, from the stochastic model component (f_N) in Equation 4 to incorporate fundamental uncertainty.

5. For each of the M sets of m simulated values, I average over the fundamental uncertainty by computing the mean of the m simulations, thus obtaining one vector of M expected values: $\mathbb{E}(Y \mid T) = \frac{1}{m} \sum_{k=1}^{m} \tilde{y}_k$.

[2] Y is regressed on T only since covariate balance is obtained through matching.

These expected values are used to visualise the uncertainty surrounding the model parameters and to compare the distributions of $\mathbb{E}(Y \mid T = 0)$ and $\mathbb{E}(Y \mid T = 1)$. First, the model is run with a continuous predictor, comparing counties with incarceration rates at one standard deviation below the global mean ($T = 0$) to those one standard deviation above the global mean ($T = 1$). The above steps are done for $T = 0$ and for $T = 1$ separately before differences in the expected values of Y are examined. As a sensitivity measure, I modify the "treatment" to be defined for a county above the 60^{th} incarceration percentile. The number of simulations is set to $M = m = 100,000$. All statistical analyses are conducted in `R`, version 3.5.1 (R Core Team, 2018), using the software packages `dplyr` (Wickham et al., 2018), `ggplot2` (Wickham, 2016), `plm` (Croissant and Millo, 2008), `MatchIt` (Ho et al., 2011), and `Zelig` (Imai et al., 2008; Choirat et al., 2018).

4.3 Findings

Figure 4.1 shows the mortality rate from drug use disorders across counties over time. There is not only a rise in such mortality rates, accelerating from the mid-1990s onwards, but also increasing between-county inequality over time. Figure 4.2 confirms a social gradient in drug-related deaths, yet highlights the existence of substantial heterogeneity across counties, notably at the bottom of the income distribution. Figures 4.3 and 4.4 show that also local admissions rates to jails and prisons are unequal across the income spectrum. When viewed in tandem with Figures 4.5 and 4.6, this indicates that penal expansion may play a role in driving heightened substance

abuse-related mortality risk in the most deprived counties and, based on the evidence reviewed in the previous chapters, in the most deprived neighbourhoods within counties. The following analysis seeks to parametrically model this hypothesis.

Table 4.3 shows results from the fixed effects regression. In this model, all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation. Moreover, the outcome variable is the natural logarithm of the age-standardised mortality rate from drug-use disorders, thus rendering coefficients interpretable as the percentage change in the mortality rate associated with a one standard deviation increase in each predictor – or in econometric terms, the semi-elasticity of Y with respect to X. The presented standard errors are estimated using a robust covariance matrix of parameters for panel models according to the Arellano version of the White method, which allows a fully general structure with respect to serial correlation and heteroskedasticity (Arellano, 1987).[3] The results suggest that both jail and prison incarceration rates are associated with elevated mortality rates from drug use disorders, net of the aforementioned control variables. A one standard deviation increase in the rate of jail admissions is associated with a 1.5% increase in drug-related deaths (95% confidence interval [CI]: [1.0, 2.0]; $P < 0.001$). The corresponding figure for prison admissions rates is 2.6% (95% CI: [2.1, 3.1]; $P < 0.001$). These associations hold over and above the effect of declining median household income, which is associated with a 12.8% rise in drug-related mortality (95% CI: [11.0, 14.6];

[3] For all results presented in this thesis, the precision of the parameter estimates is robust to the choice of standard error estimator.

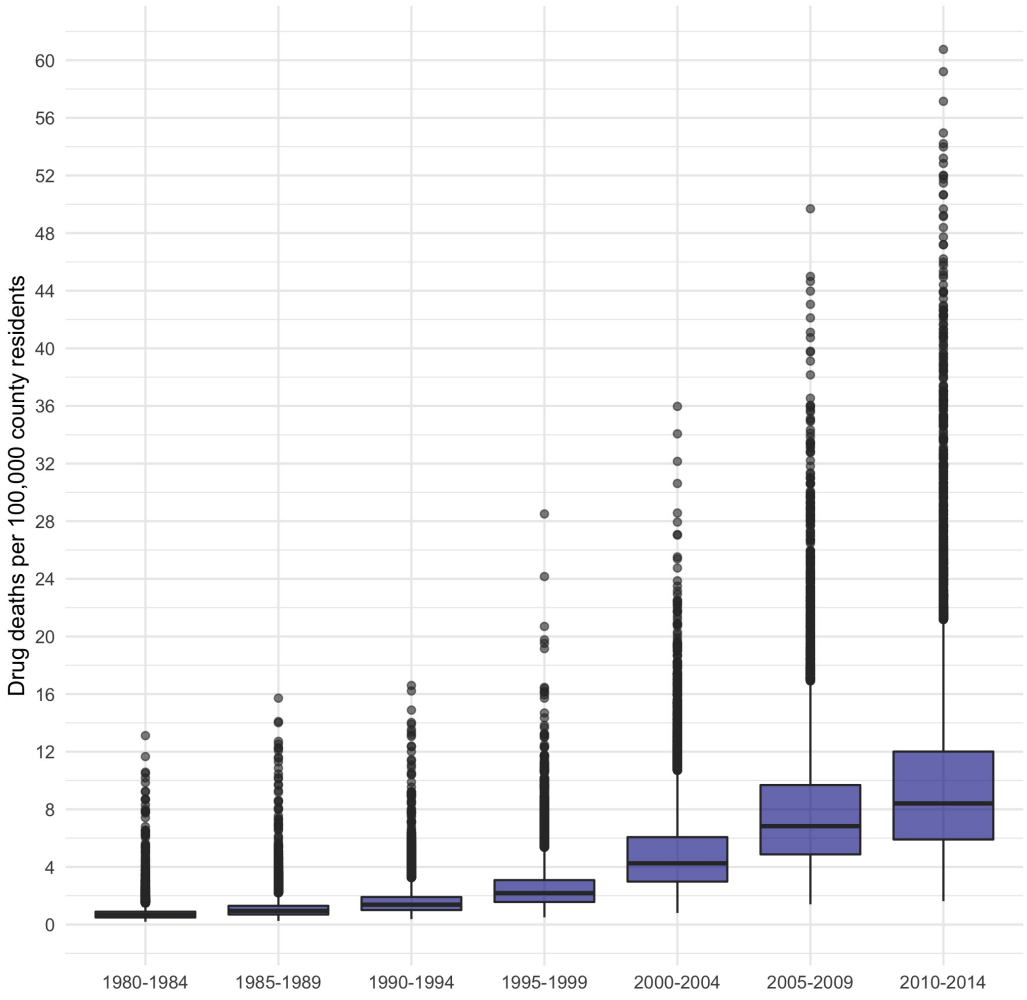

Figure 4.1: *Rise of age-standardised mortality rates from drug use disorders, 1980–2014.*

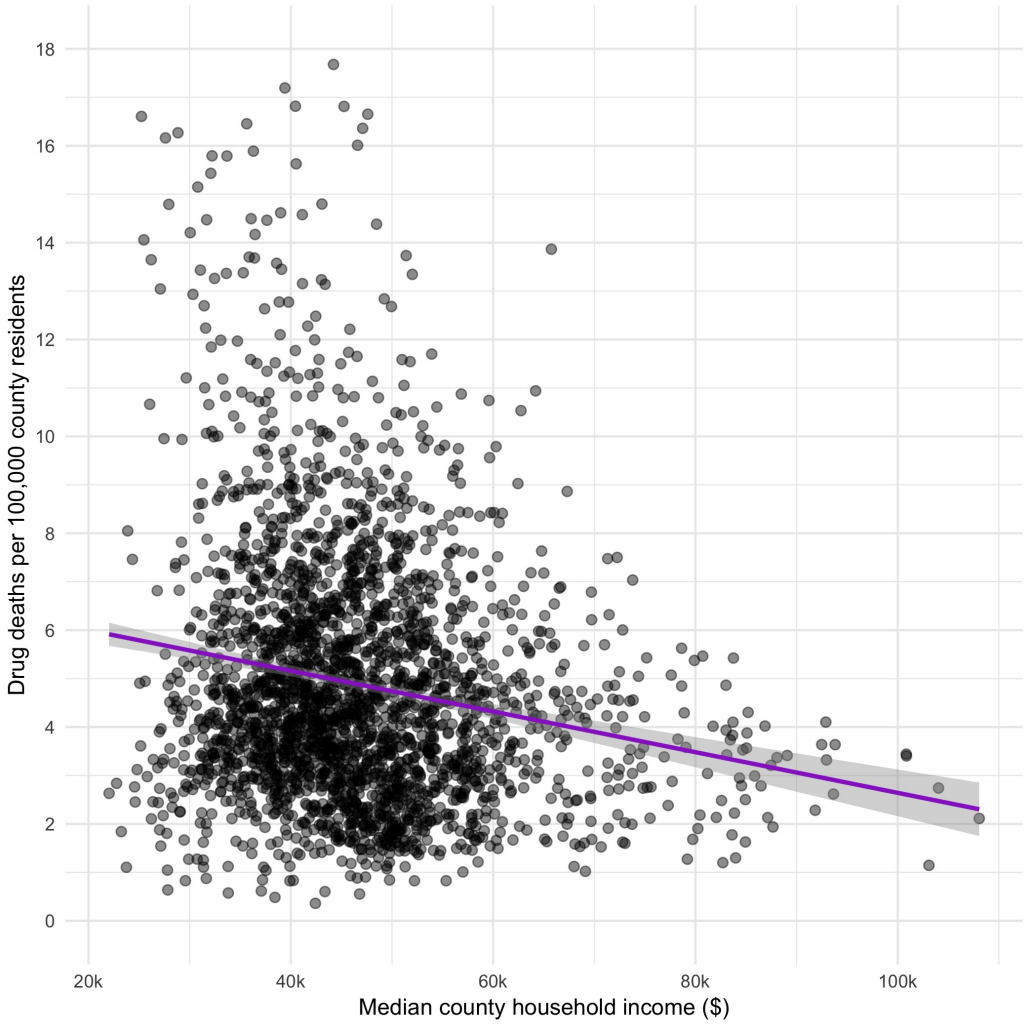

Figure 4.2: *Bivariate association between median household income and age-standardised mortality rates from drug use disorders (1980–2014 average).*

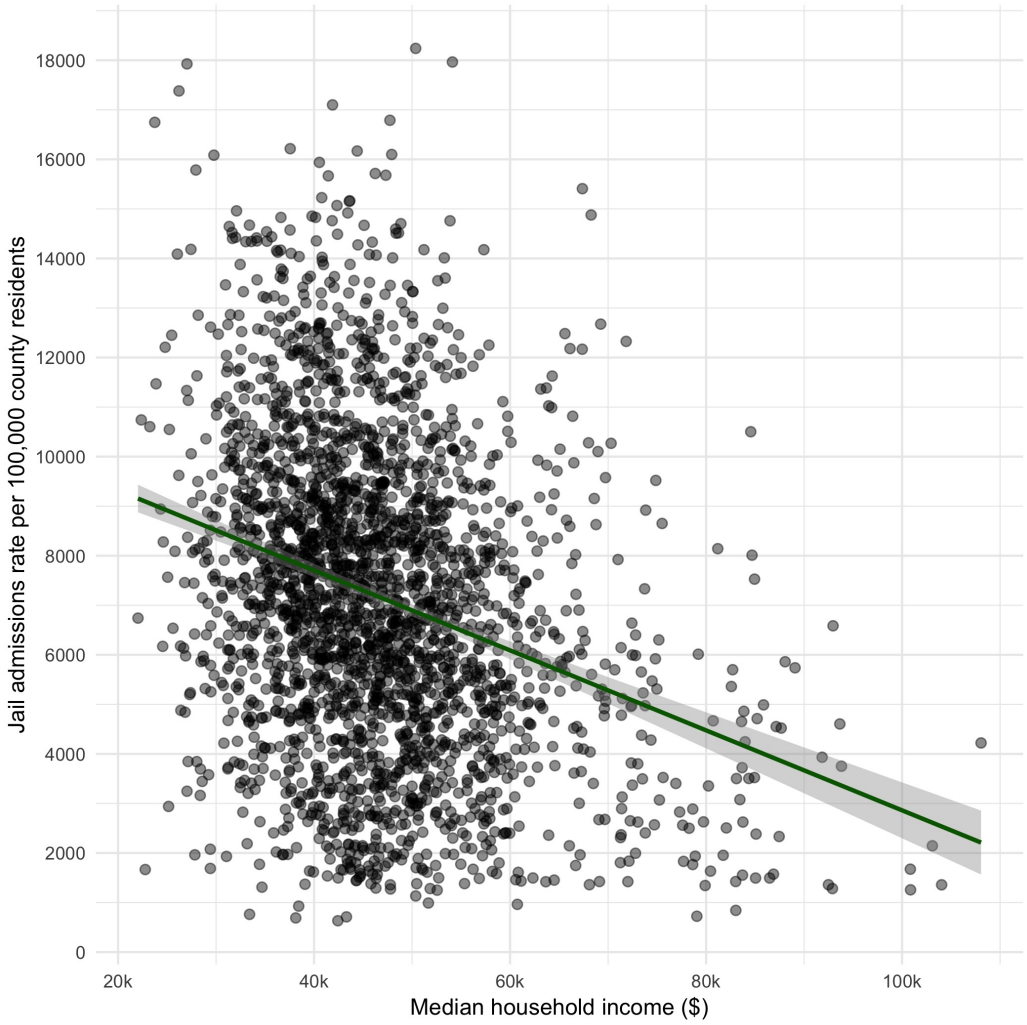

Figure 4.3: *Bivariate association between median household income and jail admissions rate (1980–2014 average).*

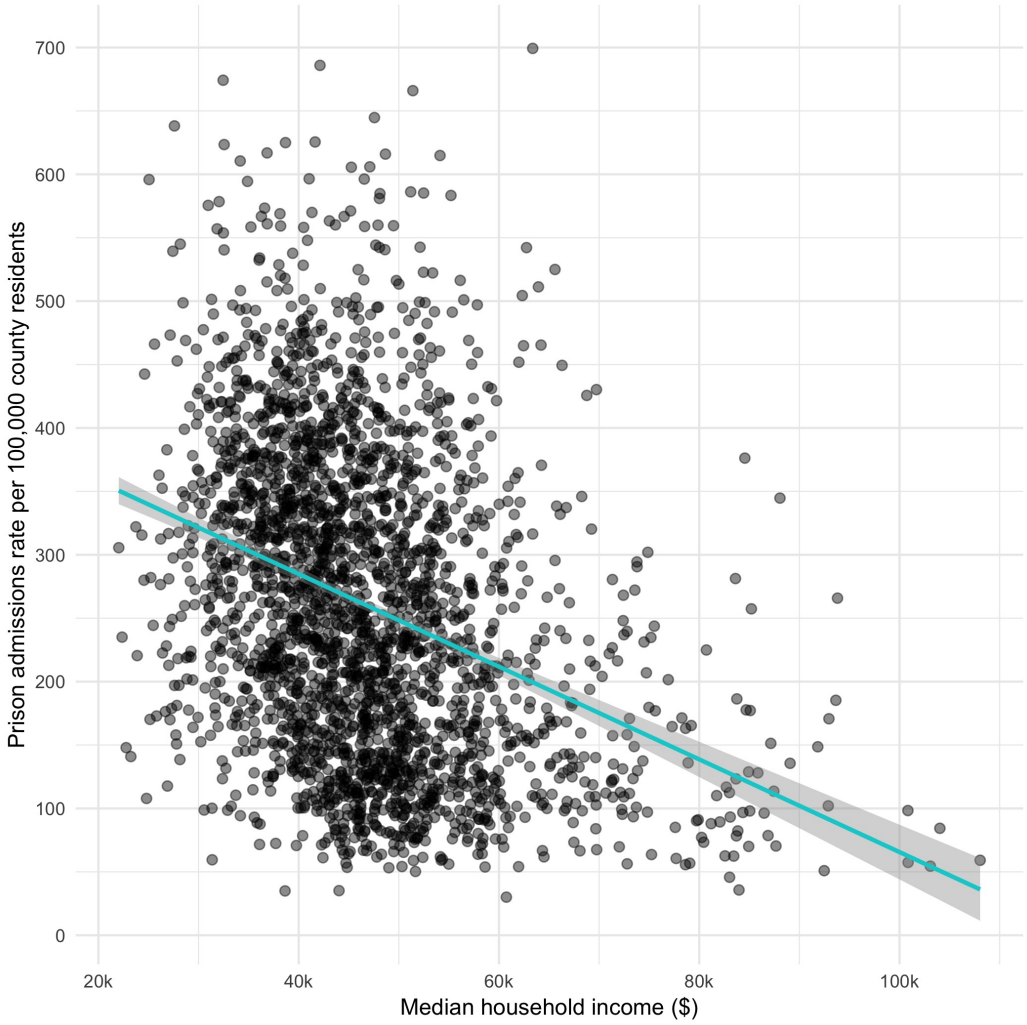

Figure 4.4: *Bivariate association between median household income and prison admissions rate (1980–2014 average).*

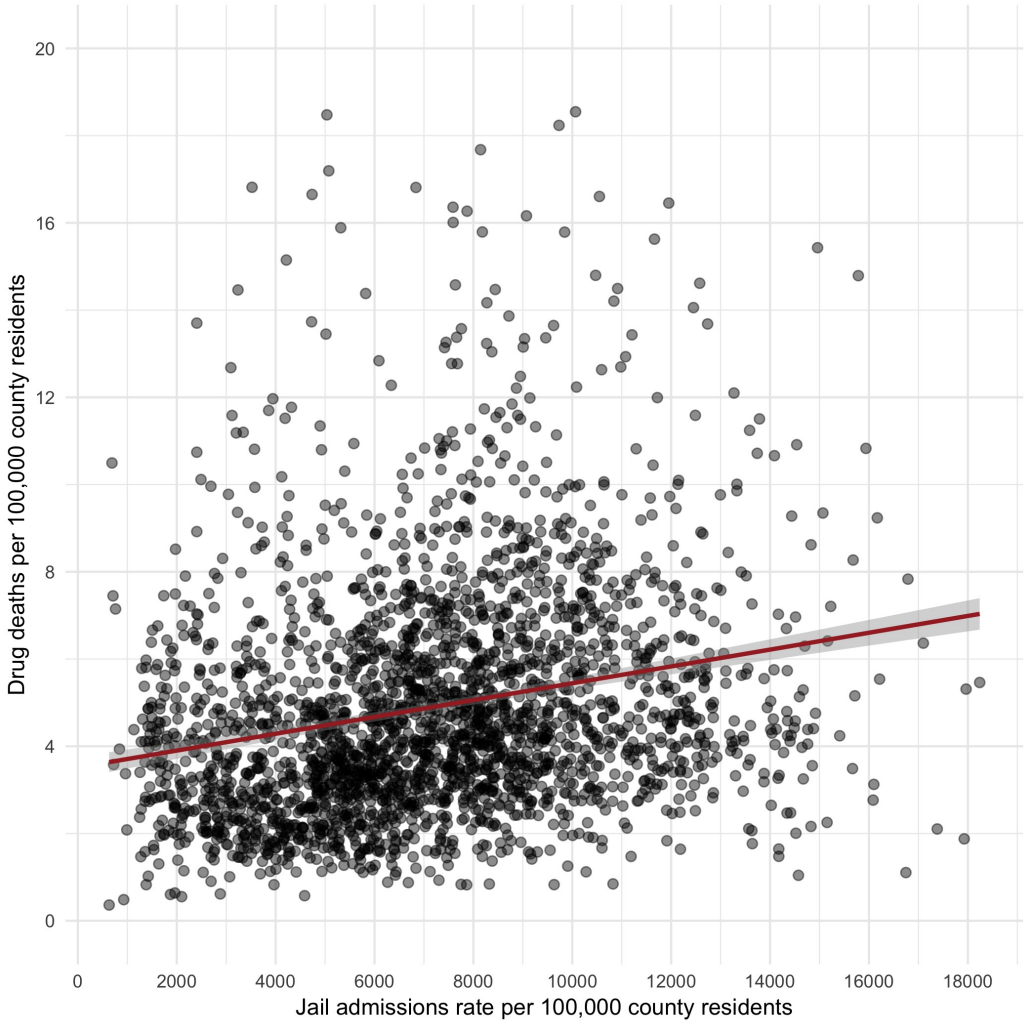

Figure 4.5: *Bivariate association between jail admissions rate and age-standardised mortality rates from drug use disorders (1980–2014 average).*

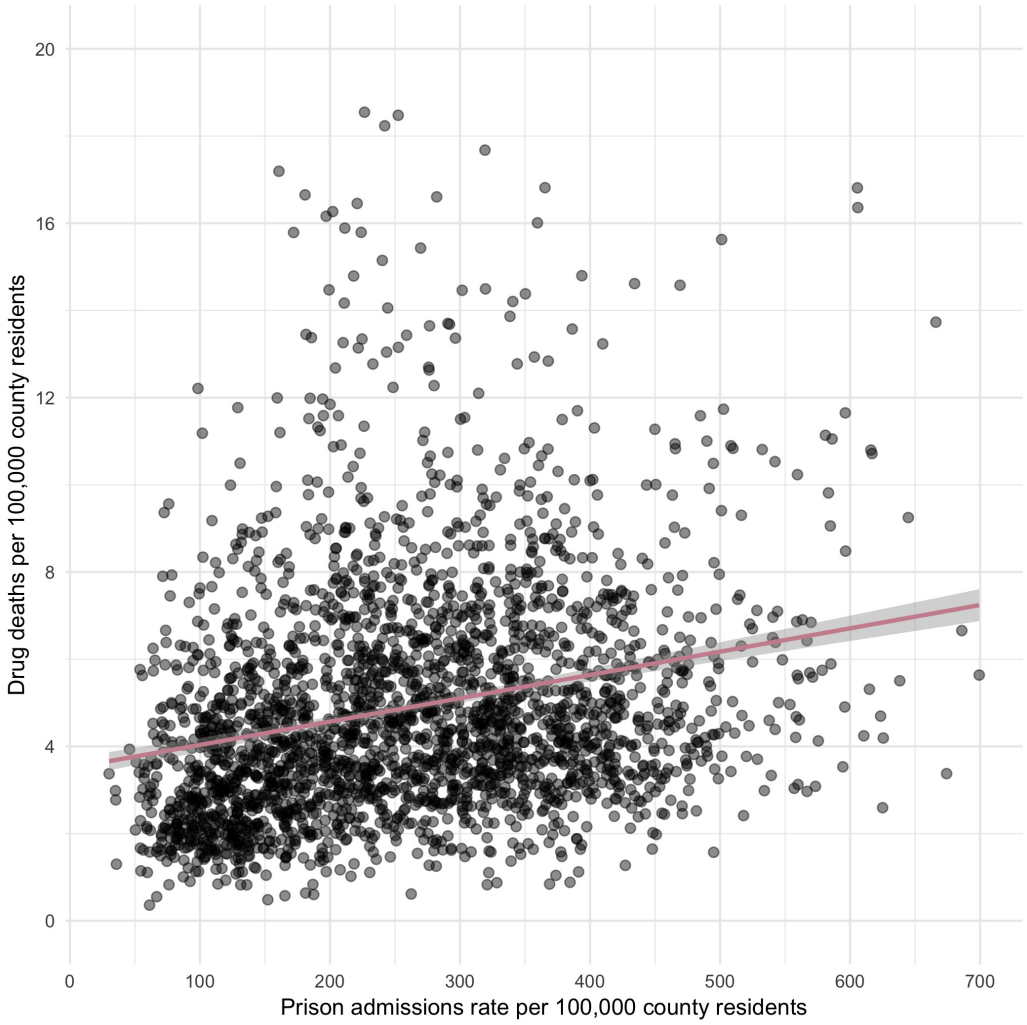

Figure 4.6: *Bivariate association between prison admissions rate and age-standardised mortality rates from drug use disorders (1980–2014 average).*

$P < 0.001$). Amongst the other regressors, violent crime is, as expected, associated with an elevated death rate from drug use disorders ($\beta = 1.0\%$; 95% CI: [0.3, 1.6]; $P < 0.01$). The remaining coefficients must be interpreted with great caution, given that within-county variation in the fraction of high school graduates or ethno-racial composition is typically small and is nearly time-invariant. These predictors will be easier to interpret when analysing between-county variation, to which I proceed below. The fixed effects model explains nearly all the within-county variation ($R^2 = 97.5\%$), although also this figure must be interpreted with caution given, once again, that a substantial proportion of variation in the outcome variable lies between rather than within counties. Nonetheless, these findings suggest a robust association between penal expansion and the overdose epidemic, notably insofar as high rates of incarceration may compound the deleterious health effects of regional economic decline.

I also find that the results are robust to controlling for all-cause mortality rates, suggesting that the findings do not merely reflect the endogenous health profile of counties. Moreover, I run a conventional one-level random effects model (not displayed), which "pools" within- and between-county variation. This model produces virtually identical substantive results. Once again, incarceration rates are associated with higher mortality rates from drug use disorders ($\beta_{jail} = 1.8\%$; 95% CI: [1.3, 2.3]; $P < 0.001$; $\beta_{prison} = 2.9\%$; 95% CI: [2.4, 3.4]; $P < 0.001$). As in the fixed effects regression, the other covariates – with the exception of reduced median household income ($\beta = 13.1\%$; 95% CI: [11.4, 14.9]; $P < 0.001$) and violent crime ($\beta = 1.5\%$; 95% CI: [0.9, 2.2]; $P < 0.001$) – produce coefficients that are difficult to interpret, but also here

a high proportion of variation in drug-related mortality is captured ($R^2 = 97.3\%$). A second type of random effects model, which explicitly partitions the total variation into variation within and variation between counties, will help shed further light on the matter below.

Next, I fit a multilevel random effects "within-between" model in order to simultaneously inspect variation within and variation between counties. Because this model more than doubles the number of estimated parameters compared to the fixed effects approach, I add and remove control variables one by one to avoid over-specification. However, in all models, I retain the year dummies to adjust for time fixed effects. A baseline model without any controls is displayed in Table 4.4. The model estimates that the within-county association between a one-standard deviation increase in jail admissions rates and drug-related mortality is equal to 2.4% (95% CI: [2.2, 2.6]; $P < 0.001$), whereas the between-county association is equal to 9.1% (95% CI: [7.3, 11.0]; $P < 0.001$). Corresponding figures for prison admissions rates are 3.8% (95% CI: [3.6, 4.0]; $P < 0.001$) and 11.3% (95% CI: [9.5, 13.2]; $P < 0.001$). This discrepancy between the two types of estimates pertains to differences in the source of variation. To quantify the amount of variation within and between counties, I calculate the so-called variance partition coefficient (VPC) from a "null" model, which simply estimates a random intercept without any regressors. This is obtained by dividing the intercept variance by the total variance (intercept variance plus residual variance) from the "null" model. The VPC is equal to 0.245, suggesting that no less than a quarter of the total variation lies between rather than within counties. This confirms the utility of the "within-between" specification. Comparing

Table 4.3: Fixed effects panel regression model of drug-related mortality rates

	Coefficient	Standard error	P-value
Jail admissions rate	1.5%	0.3	< 0.001
Prison admissions rate	2.6%	0.2	< 0.001
Household income decline	12.8%	0.9	< 0.001
Fraction high school graduates	5.7%	1.0	< 0.001
Fraction African Americans	-6.6%	2.8	0.02
Fraction Hispanics	-24.9%	2.0	< 0.001
Fraction other ethnicity	-14.9%	2.8	< 0.001
Violent crime rate	1.0%	0.3	0.004

Notes: The outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model controls for decline in median county household income, the county fraction of high school graduates, African Americans, Hispanics, or other non-White ethnicity, the county violent crime rate, and for aggregate annual time trends using year dummies (not displayed); robust panel-corrected standard errors are presented in the second column; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the drug-related mortality rate associated with a one standard deviation increase in each predictor. $N = 57,732$. $R^2 = 97.5\%$.

Table 4.4: Baseline multilevel random effects "within-between" panel regression

		Coefficient	Standard error	P-value
Jail admissions rate				
	Within	2.4%	0.1	< 0.001
	Between	9.1%	0.9	< 0.001
Prison admissions rate				
	Within	3.8%	0.1	< 0.001
	Between	11.3%	0.9	< 0.001

Notes: The outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model only controls for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the drug-related mortality rate associated with a one standard deviation increase in each predictor. $N = 64,814$.

the baseline model to the "null" model reveals that the addition of both incarceration predictors reduces the intercept (between-county) variance by almost 20% and the residual (within-county) variance by 97%.

Figure 4.7 visualises the parameter estimates and corresponding 95% confidence intervals of each of the control models. Each panel shows the coefficients for jail and prison admissions rates, adjusted for the control variable whose label appears on the right hand side (as well as for aggregate time

trends). For instance, the first panel (from the top) displays the association between incarceration and age-standardised mortality rates from drug use disorders, net of the effect of reduced median household income at the county level. The second panel does the same for the county fraction of high school graduates, and so on. The overall distribution of coefficients confirms that the "within" estimator may produce extreme results,[4] notably in the case of variables that are mostly time-invariant within counties, whereas the "between" estimates lend themselves to easier interpretation. Thus, as expected, counties with a higher fraction of high school graduates have *lower* rates of drug-related mortality. Similarly, the coefficients for the ethno-racial composition of counties are more meaningful when looking at differences between rather than changes within counties. The association between a concentration of African American residents and death rates from drug use disorders is reduced (but still negative), for Hispanics it is rendered statistically insignificant, and for other ethnic minorities, it reverses sign.[5]

Figure 4.8 reproduces the same figure but without the "within" coefficients

[4] The reason that the "within" estimates produced here differ from those of the fixed effects model is that, as mentioned, the multilevel approach also models a random intercept (which the fixed effects approach suppresses). A one-level random effects "within-between" model would generate "within" estimates that are identical to that of the fixed effects approach (see Bell and Jones, 2015).

[5] It is worth noting that these coefficients cannot be interpreted as to whether belonging to a given ethnic minority entails a mortality (dis)advantage at the level of the individual. The variables in question are simply measuring the aggregate ethno-racial composition of counties, which provides limited information about the racialised patterning of vital inequality.

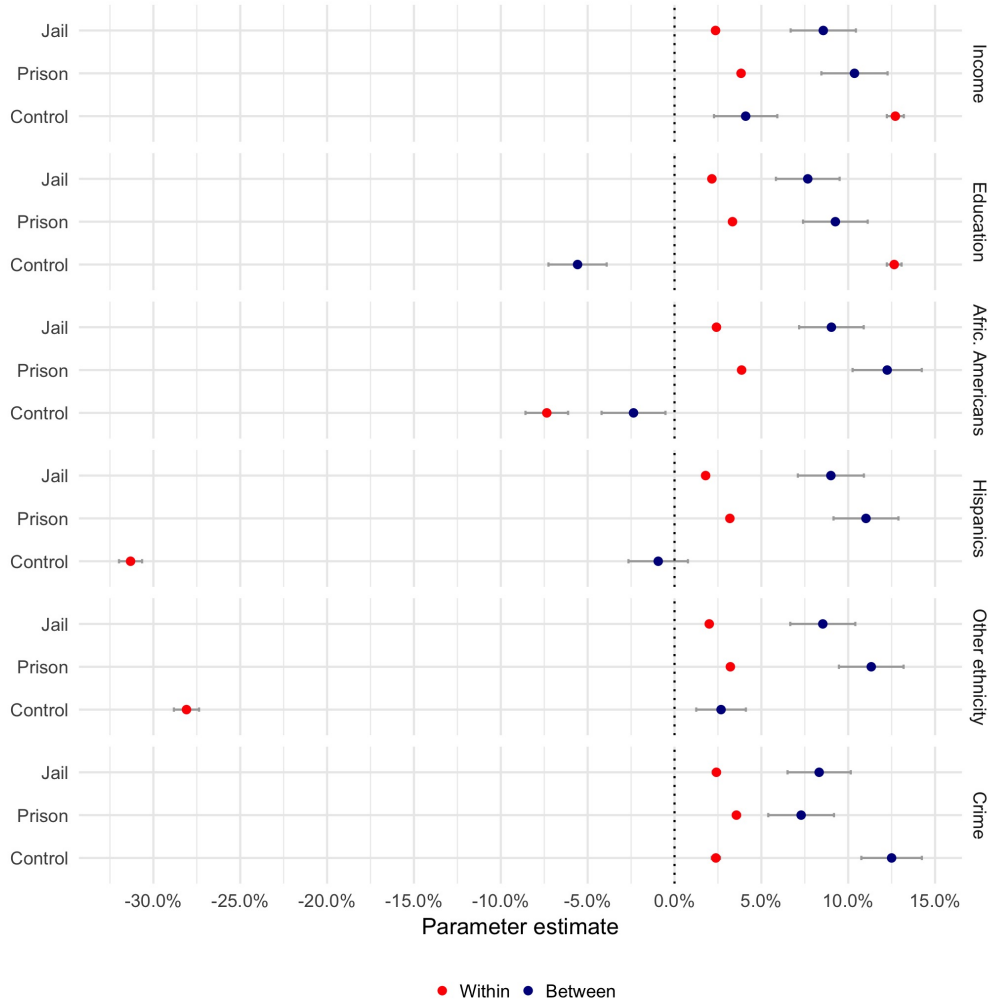

Figure 4.7: *Coefficient plot of multilevel "within-between" random effects models. The outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the main predictors are the county jail and prison admissions rates per 100,000 population, adjusted for aggregate annual time trends using year dummies; control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the drug-related mortality rate associated with a one standard deviation increase in each predictor.*

of the latter two groups, which may be considered unreliable outliers. This figure allows for more careful inspection of the main coefficients of interest and the degree of uncertainty associated with them by zooming in on a subset of the coefficient plot. The reader will note that in all models, jail and prison incarceration rates are positively associated with drug-related deaths. The "within" estimates are remarkably similar across all specifications, whereas the "between" estimates are somewhat more variable. However, the latter tend to be larger in size than the former, as is to be expected – with a few notable exceptions, such as median household income. Overall, the "within-between" approach sheds additional light on the association between penal expansion and the American overdose epidemic. It confirms the substantive findings of the relatively conservative fixed effects approach, but highlights the importance of also taking between-county variation into account. Consequently, I proceed to isolating and further probing between-county inequalities in drug-related mortality rates using the matching and simulation-based approaches described above.

The matching algorithm results in a pruned data set composed of $N = 963$ counties. Counties are matched on the following time-averaged (1980–2014) variables: median household income, the fraction of high school graduates, African Americans, Hispanics, or other non-White ethnicity, the violent crime rate, and the opioid prescription rate (2006–2014). The diagnostics reveal a high degree of balance improvement since the empirical covariate distributions in both the "treatment" and "control" groups are now similar, meaning the much reduced sample size strengthens rather than undermines the subsequent statistical inference. Hence, I proceed to estimating a "between"

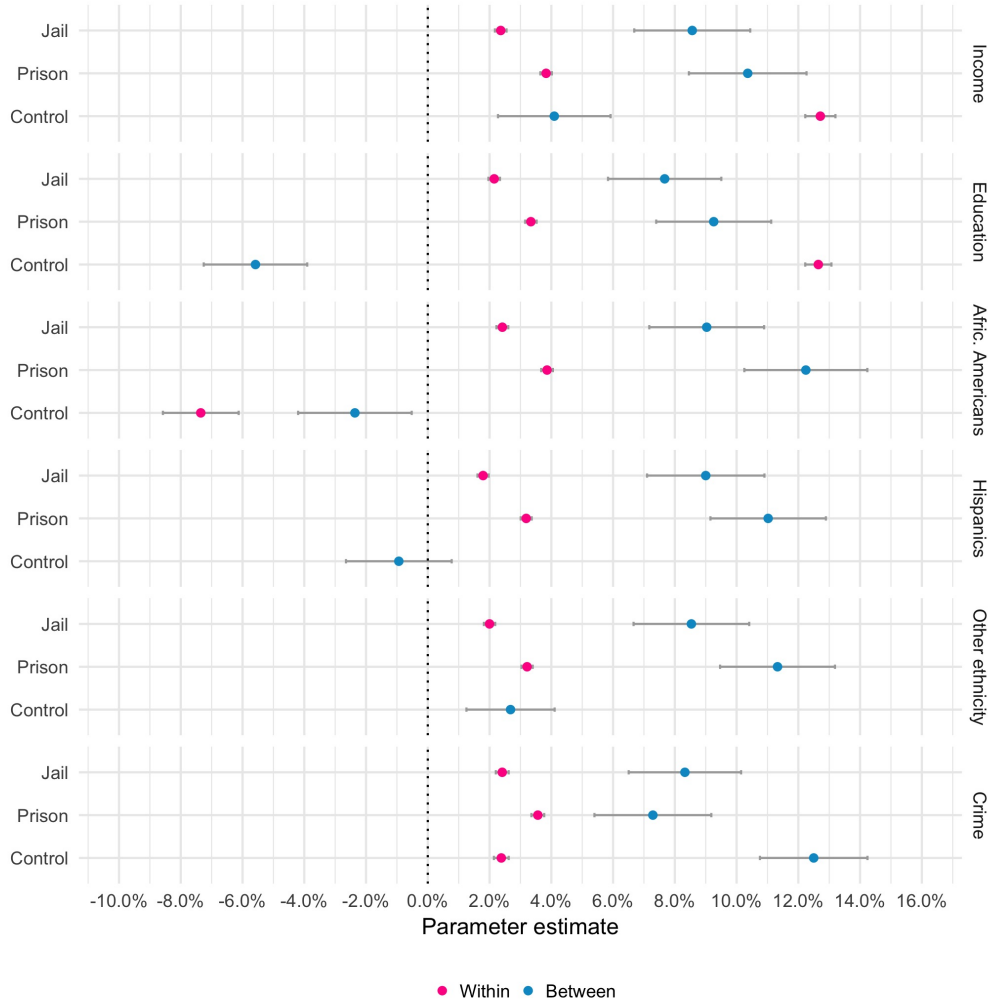

Figure 4.8: *Coefficient plot of multilevel "within-between" random effects models (without outliers). The outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the main predictors are the county jail and prison admissions rates per 100,000 population, adjusted for aggregate annual time trends using year dummies; control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the drug-related mortality rate associated with a one standard deviation increase in each predictor.*

model using simple linear regression, where the outcome, Y, is regressed on T. In this model, I also integrate data on variation in opioid prescription rates at the county level between 2006 and 2014. The model is visualised in Figure 4.9. It compares counties with incarceration rates at one standard deviation below the global mean ($T = 0$) to those with incarceration rates at one standard deviation above the global mean ($T = 1$). The model simulates counterfactuals by comparing counties with and without the presence of high rates of incarceration. In other words, to use the language of clinical trials, how does the mortality rate from drug use disorders change when comparing a county under "treatment" to one in the "control" group, (most) other things being equal?

I first estimate the expected value of deaths from drug use disorders for a county that is in the "control" group, meaning it has an incarceration rate at one standard deviation below the global mean. According to the model, as seen in the first density plot (in green), its mortality rate from drug use disorders lies at 3.5 deaths per 100,000 county residents (95% CI: [3.3, 3.7]; $P < 0.001$). Next, I estimate the expected value of the mortality rate for a county that only differs from the first in that it has incarceration rates that lie at one standard deviation *above* the global mean. As evidenced by the second (black) density plot, this raises the mortality rate from 3.5 to 5.4 deaths per 100,000 county residents (95% CI: [5.2, 5.6]; $P < 0.001$). As conveyed by the third and final density (in dark pink), which shows the first difference in the expected value of the mortality rate for the different treatment states, these results suggest that, on average, the experience of high incarceration rates corresponds to 1.9 excess deaths per 100,000 county residents (95% CI: [1.5,

2.2]; $P < 0.001$). In terms of the semi-elasticity of Y with respect to T, this result suggests a "treatment effect" equal to a 53.5% increase in the death rate from drug use disorders.

As one possible robustness check, I modify the matching algorithm to split counties into alternative "treatment" states. In this specification, T is set to 1 for any county above the 60^{th} percentile of county jail or prison incarceration, and to 0 otherwise. I then pre-process the data once more using coarsened exact matching. The substantive quantities of interest remain similar, as conveyed by Figure 4.10. Counties in the "control" group have an average mortality rate from drug use disorders equal to 4.1 deaths per 100,000 population (95% CI: [3.9, 4.3]; $P < 0.001$), whereas counties with high incarceration rates have an average drug-related mortality rate equal to 5.5 deaths per 100,000 population (95% CI: [5.2, 5.7]; $P < 0.001$). This amounts to 1.4 excess deaths (95% CI: [1.0, 1.7]; $P < 0.001$), which is equivalent to a 32.9% increase in drug-related mortality rates associated with high rates of incarceration.

4.4 Discussion

The findings presented above suggest a strong association between the rise of the penal state and the ongoing overdose epidemic. The results seem to hold over and above the links between economic decline, the local ethnoracial composition, the drug environment, or crime rates. This chapter also highlights a largely neglected dimension of the American criminal justice system, namely local jails, which are independently associated with drug-related

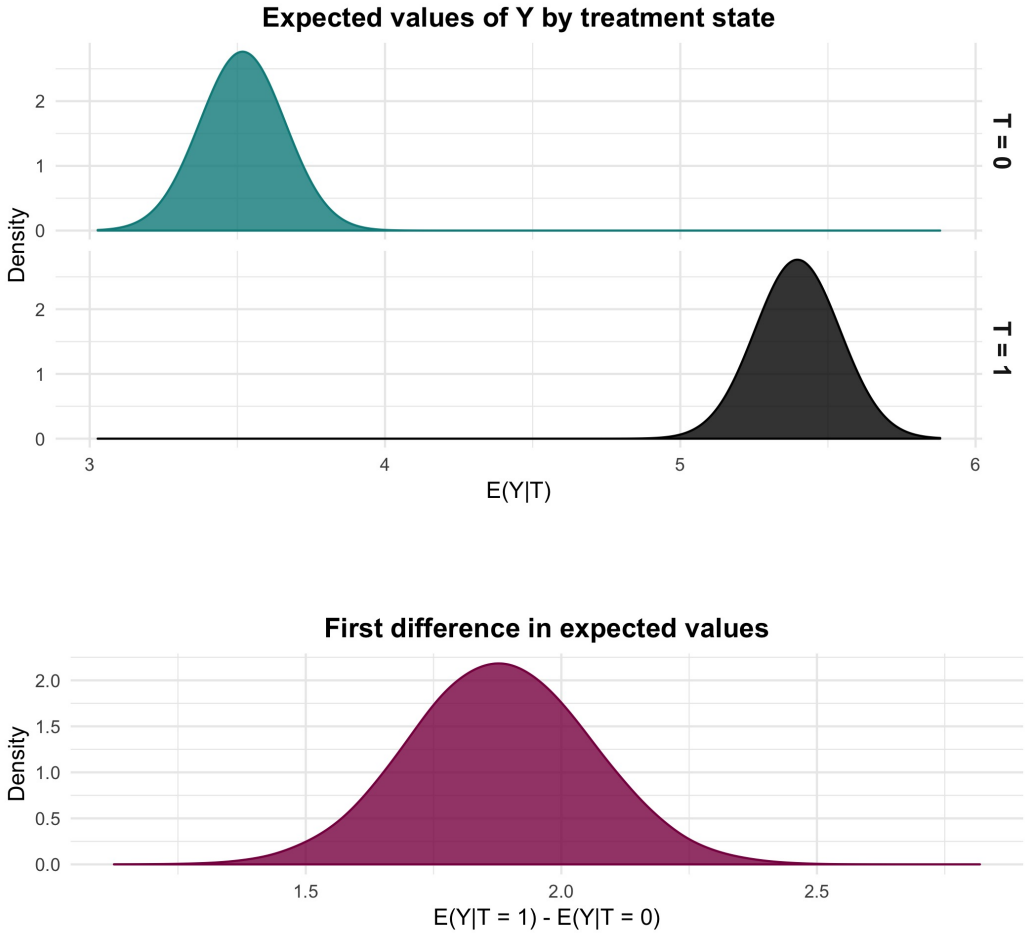

Figure 4.9: *Density plots of simulated expected values of drug-related mortality given a "treatment" (T) state. The outcome variable is the age-standardised mortality rate from drug use disorders; the model compares counties with incarceration rates at one standard deviation below the global mean ($T = 0$) to those with incarceration rates at one standard deviation above the global mean ($T = 1$); the association between drug-related mortality and "treatment" is estimated by applying a simple linear regression model to a pruned data set that is pre-processed using coarsened exact matching. $N = 963$.*

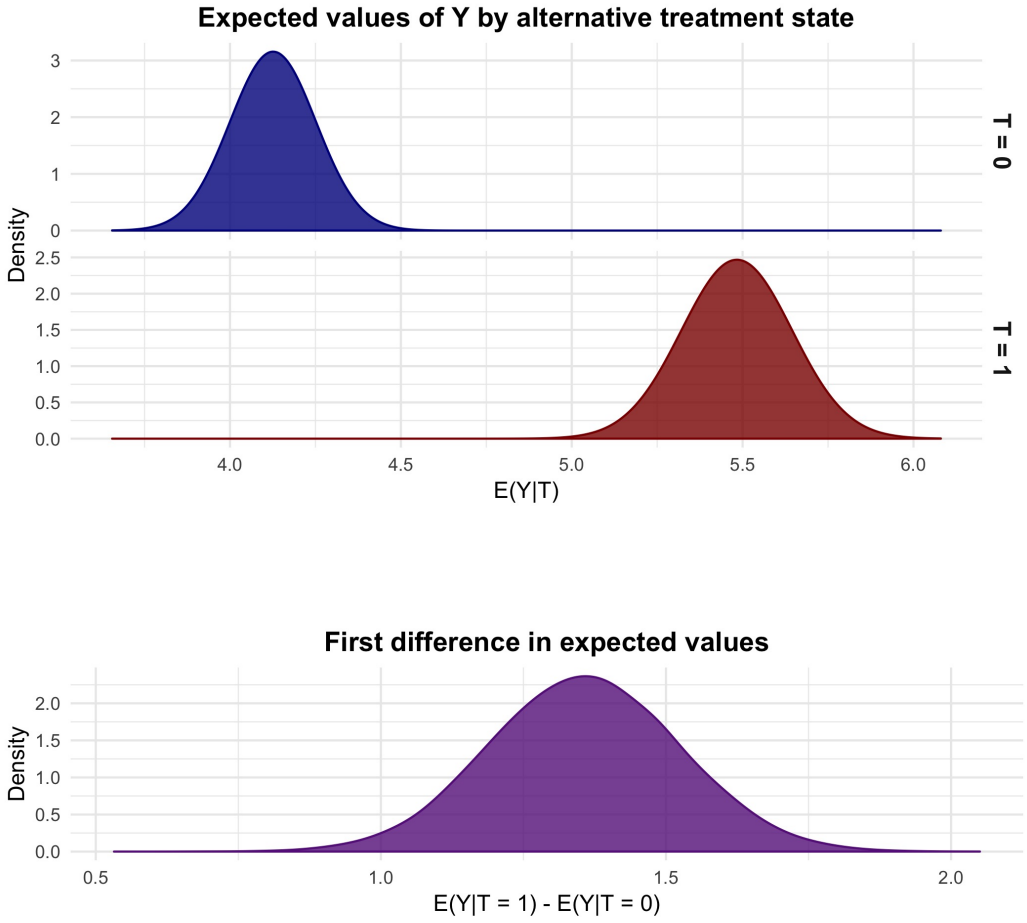

Figure 4.10: *Density plots of simulated expected values of drug-related mortality given a "treatment" (T) state. The outcome variable is the age-standardised mortality rate from drug use disorders; "treatment" is defined as above the 60^{th} percentile of county jail or prison incarceration rates; the association between drug-related mortality and "treatment" is estimated by applying a simple linear regression model to a pruned data set that is pre-processed using coarsened exact matching; counties are matched on the same variables as in Figure 4.9 above. $N = 989$.*

deaths. The use of panel data sheds light on regional inequalities over time and thus helps unpack the potential directionality of relevant associations between criminal justice and substance abuse that have been identified in a recent cross-sectional study of individuals (Winkelman et al., 2018).

I acknowledge the limitations of this analysis. First, there is the question of whether the link between incarceration and drug-related mortality is subject to selection bias and thus whether the impact of incarceration *per se* has been statistically identified (see for instance Johnson and Easterling, 2012; Wildeman et al., 2013). In any observational study, there is always a concern that an observed association can be explained away by some unaccounted-for third factor related to both the treatment and the outcome. However, considerable unmeasured confounding would be needed to explain away the model estimates presented above, which are adjusted for the most likely confounders in the American context. The fixed effects model eliminates any time-invariant confounders within counties, whilst the matching procedure helps balance covariates distributions between counties. Those who face incarceration do indeed have different health profiles compared to the general population (National Research Council, 2014: 204–213), but the transition from poor health to actual mortality seems to be accelerated by the experience, first-hand or vicarious, of penal confinement. The interaction between drug abuse and incarceration interferes with treatment and reduces the likelihood of recovery (Fiscella et al., 2004; Nunn et al., 2009; National Research Council, 2014: 217–219; Maradiaga et al., 2016). Moreover, the above analysis suggests that jails and prisons are associated with heightened mortality risk for the population at large, and not only for those individuals who are

most directly affected by the carceral apparatus. Finally, as outlined in chapter 3, existing research on the effects of incarceration on a range of social, economic, and health outcomes lends credence to my principal hypothesis that punitive social policy, notably in the wake of economic decline, is a likely driver of regional variation in drug-related mortality.

Second, the analysis could have profited from better data. A multilevel framework in which individuals are nested in neighbourhoods, which in turn are nested in cities, counties, and states, would allow for a more refined examination of the associations at work and would also help better address the aforementioned concern surrounding endogeneity. As mentioned earlier (see Figure 8 in the Introduction), incarceration only constitutes one part of a broader set of operations of the penal state and there are currently no consistent data collections that would allow for a systematic analysis of these, including probation and parole. Nonetheless, although my data set does not cover all states, it contains empirical data on jail and prison incarceration rates at the county level for the very first time. My measurement of income decline is at the aggregate level and would ideally be complemented by a more comprehensive set of economic indicators, including import-export flows, labour force participation rates, and employment dynamics (see e.g. Pierce and Schott, 2016b; Ruhm, 2018).

Third, partly as a consequence of these data limitations, the statistical models presented above cannot do justice to the causal complexity at work, as visualised in Figure 3.1. Most notably, in light of the socially differentiated operations of the penal state, an examination of heterogeneous "treatment effects" and mediating pathways across diverse sociospatial and

temporal dimensions would be of particular interest. However, in the absence of individual-level data and more refined measures of likely mediators and moderators, I have refrained from such an endeavour in the present analysis. The different model specifications yield results of differing magnitudes, but these pertain to differences in the distribution of variation within and between counties, and can be considered reasonable bounds on the magnitude of potential relationships. The parameter estimates are largely consistent across the different models, my substantive findings are robust to alternative specifications, and the parsimonious models successfully capture a substantial portion of the variation in the data.

4.5 Conclusion

There is a need to understand why drug-related mortality rates are subject to substantial regional variation if roadmaps to intervention and prevention are to be developed. The findings of this chapter suggest that incarceration may be an important upstream determinant of mortality from drug use disorders in the United States, over and above the effects of economic decline and other usual suspects. Jails and prisons are associated with heightened mortality risk, not only for people sent there but for the population at large. The rapid expansion of the penal state over the past few decades may thus have contributed significantly to the ongoing wave of drug-related deaths.

Chapter 5

Penal expansion, mortality, and life expectancy

5.1 Introduction

The previous chapter demonstrated a robust association between high rates of jail and prison incarceration and higher rates of mortality from drug use disorders. This chapter expands the empirical lens to ask whether penal expansion can help explain broader patterns of vital inequality. As described in chapter 3, it is reasonable to hypothesise that the macro-level forces of which rapid penal expansion is a prominent manifestation shape a wide gamut of health outcomes via a complex set of interlocking mechanisms. In this chapter, I examine three forms of vital inequality: inequalities in age-standardised all-cause mortality rates, inequalities in the risk of premature death between the ages of 25 and 45, and inequalities in life expectancy at birth. Figures 5.1, 5.2, and 5.3 show the evolution of these three variables at the county level

between 1980 and 2014. The all-cause mortality rate and life expectancy at birth have improved on average over time, but inequalities have increased. For instance, the healthiest counties have gained around 6 years of life expectancy in the given time period, whereas the unhealthiest have gained around 4 years. The life expectancy gap has gone from around 15 years in 1980 to almost 20 years in 2014. The premature mortality risk between the ages of 25 and 45 has moderately improved, but the most disadvantaged counties have not experienced any drastic reduction in such risk over the past three and a half decades.

5.2 Empirical strategy

Data

My empirical strategy in this chapter is similar to that of chapter 4. I use data from the Institute for Health Metrics and Evaluation (Dwyer-Lindgren, 2016; 2017) to measure county-level age-standardised all-cause mortality rates, the risk of premature death between the ages of 25 and 45, and life expectancy at birth. Descriptive statistics for these variables are presented in Table 5.1. The regressors are the same as in the previous chapter (see Table 4.1). Table 5.2 shows the Spearman correlations between all the variables used in the analysis. The reader will note the perfect inverse correlation between all-cause mortality rates and life expectancy at birth. The reason I include both outcomes in this chapter is that life expectancy lends itself to easier, more intuitive interpretation (few people think in terms of age-standardised all-cause mortality rates). However, as noted by Case and Deaton (2017: 400),

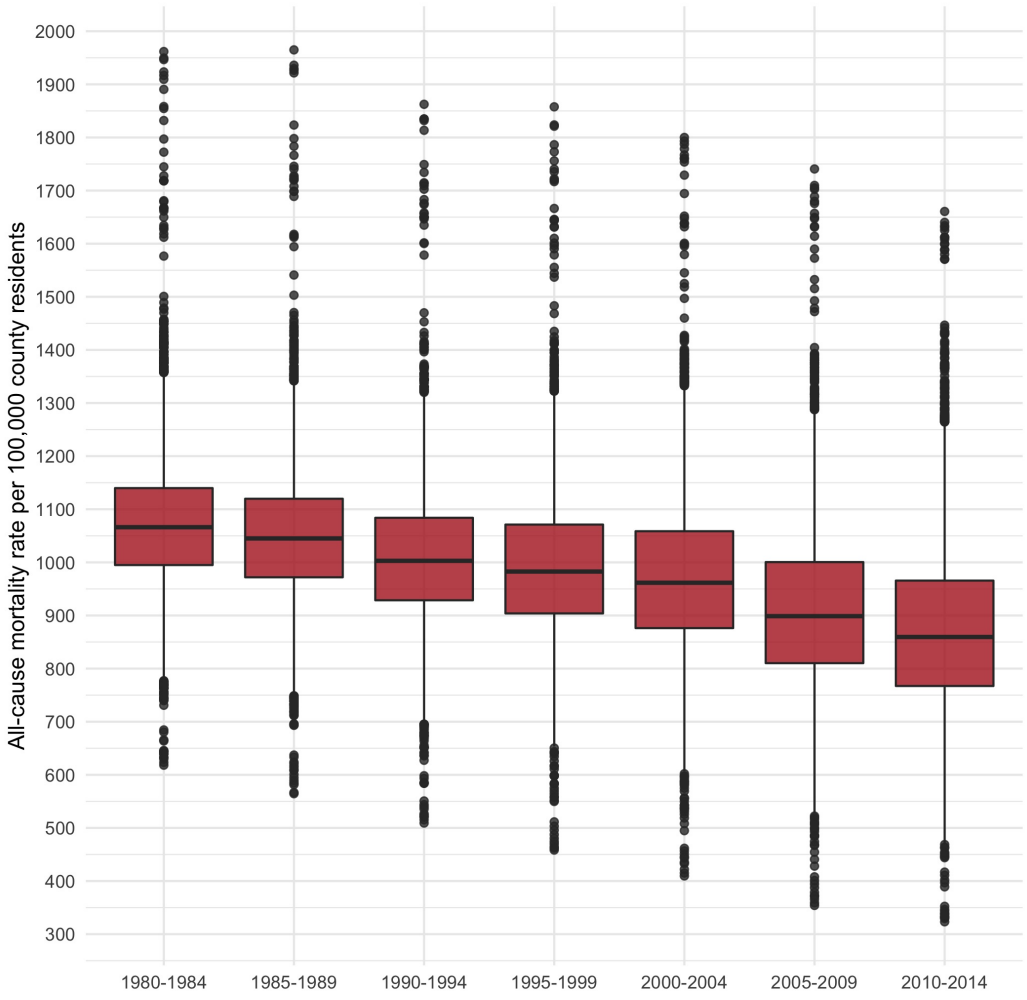

Figure 5.1: *Evolution of age-standardised all-cause mortality rates, 1980–2014.*

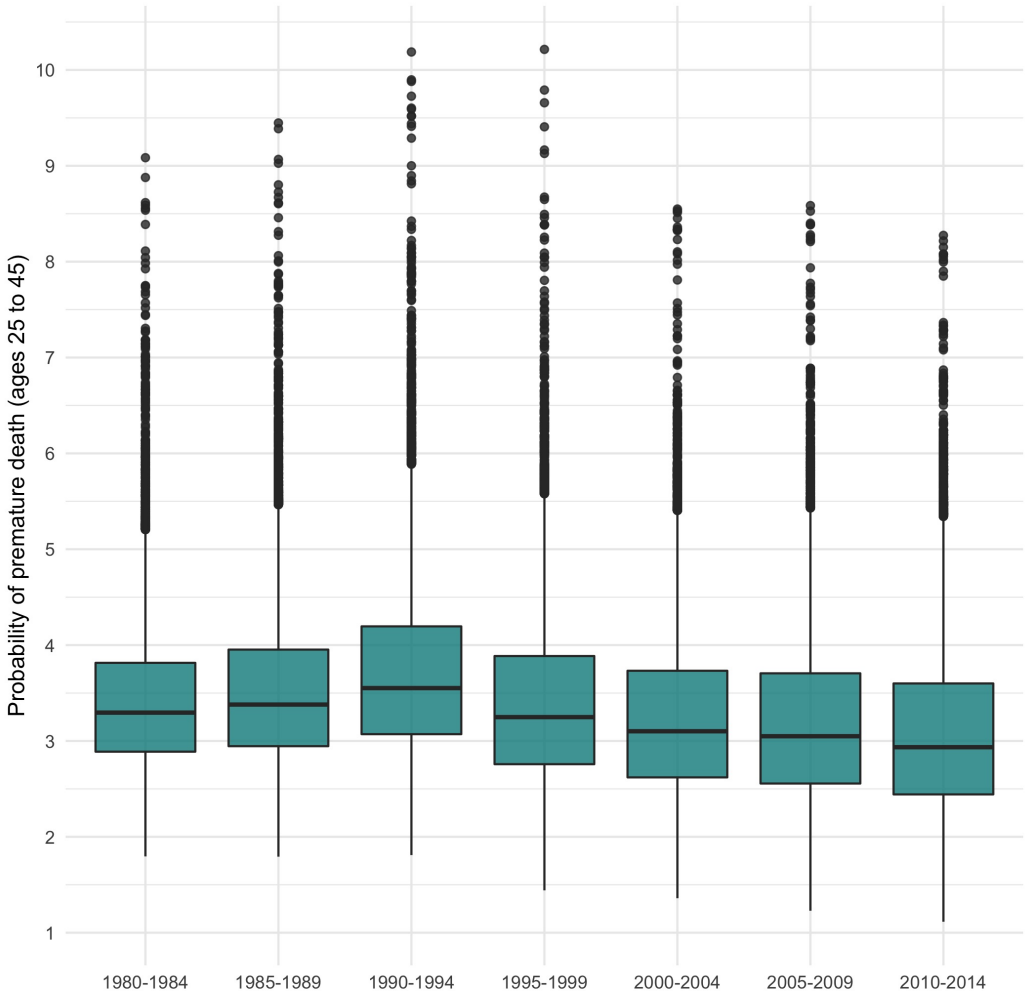

Figure 5.2: *Evolution of premature mortality risk, ages 25 to 45, 1980–2014.*

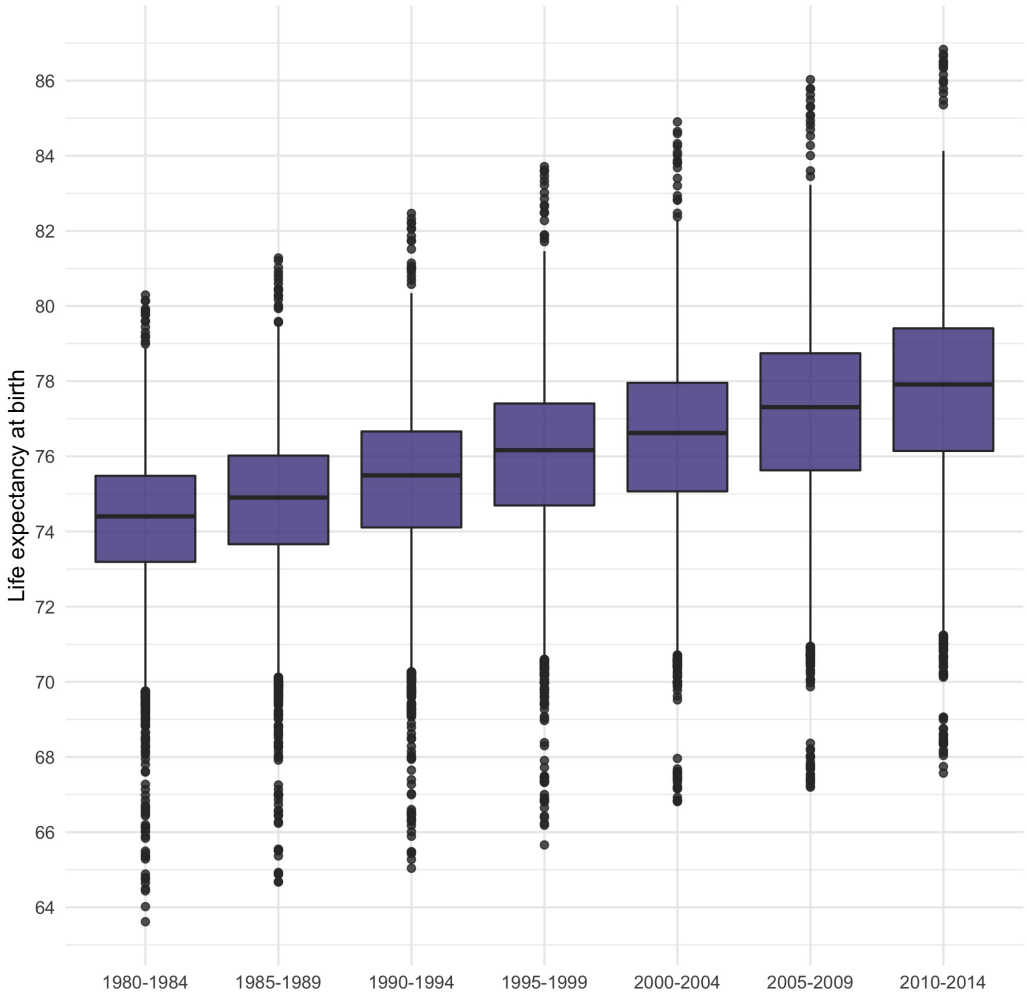

Figure 5.3: *Evolution of life expectancy at birth, 1980–2014.*

Table 5.1: Descriptive statistics

Statistic	N	Mean	St. Dev.	Min	Max
All-cause mortality rate	69,562	980.0	138.4	323.3	1,716.8
Premature mortality risk	69,562	3.4	0.9	1.2	10.2
Life expectancy at birth	69,562	76.1	2.3	66.2	86.8

Notes: Rates are per 100,000 county residents; the all-cause mortality rate is age-standardised; the premature mortality risk measures the probability of death between the ages of 25 and 45, and it is interpreted as a percentage probability.

life expectancy is more sensitive to child mortality compared to midlife or old-age mortality, and may thus shroud heterogeneity across the life course. This is why, in addition to age-standardised all-cause mortality, I include measures of premature mortality in the 25–45 age range – which is also a demographic that is central to the operations of the penal state. The reader will note, from reading Table 5.1, that variation in the probability of premature death is substantial, ranging from around 1 in 100 residents to over 1 in 10 residents.

Methods

My methodological approach is the same as in the previous chapter (see section 4.2). For each of the three outcomes, I estimate a fixed effects panel regression. I then partition the within- and between-county variation using multilevel random effects models, which I fit using one control variable at

Table 5.2: Spearman correlation matrix

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. All-cause mortality rate	1											
2. Premature mortality risk	0.8	1										
3. Life expectancy at birth	-1.0	-0.9	1									
4. Jail admissions rate	0.1	0.3	-0.2	1								
5. Prison admissions rate	0.2	0.4	-0.2	0.4	1							
6. Median household income	-0.5	-0.7	0.6	-0.2	-0.2	1						
7. Fraction high school graduates	-0.7	-0.7	0.8	-0.1	-0.1	0.6	1					
8. Fraction African Americans	0.4	0.5	-0.4	0.2	0.4	-0.2	-0.2	1				
9. Fraction Hispanics	-0.4	-0.2	0.3	0.2	0.3	0.2	0.3	0.2	1			
10. Fraction other ethnicity	-0.4	-0.3	0.4	0.1	0.1	0.3	0.5	0.02	0.6	1		
11. Violent crime rate	0.2	0.3	-0.3	0.3	0.3	-0.01	-0.1	0.5	0.3	0.2	1	
12. Opioid prescription rate	0.4	0.4	-0.4	0.3	0.3	-0.3	-0.2	0.2	-0.1	-0.1	0.2	1

a time. To further investigate between-county inequalities, I once again use coarsened exact matching to obtain covariate balance before regressing each outcome variable on a "treatment" variable. As before, I compare counties at one standard deviation below the global incarceration average to those at one standard deviation above the average.

5.3 Findings

5.3.1 All-cause mortality

Figure 5.4 shows a clear bivariate association between median household income and all-cause mortality rates at the county level. This association is even more distinct than the one between income and mortality rates from drug use disorders, as visualised in Figure 4.2 in the previous chapter. Figures 5.5 and 5.6 further suggest that incarceration may be contributing to this association, notably in light on the strong link between poverty and penal confinement (see Figures 4.3 and 4.4).

Table 5.3 shows the results of the fixed effects panel regression for all-cause mortality rates. A one standard deviation increase in jail and prison admissions rates is associated with a 0.5% (95% CI: [0.4, 0.7]; $P < 0.001$) and a 1.2% (95% CI: [1.1, 1.3]; $P < 0.001$) increase in all-cause mortality rates, respectively. These estimates remain virtually identical when additionally controlling for the local mortality rate from drug use disorders. This suggests that the model is not simply capturing the effects of the overdose epidemic, as was documented in the previous chapter. In other words, penal expansion seems to affect the broader dynamics of vital inequality, as pro-

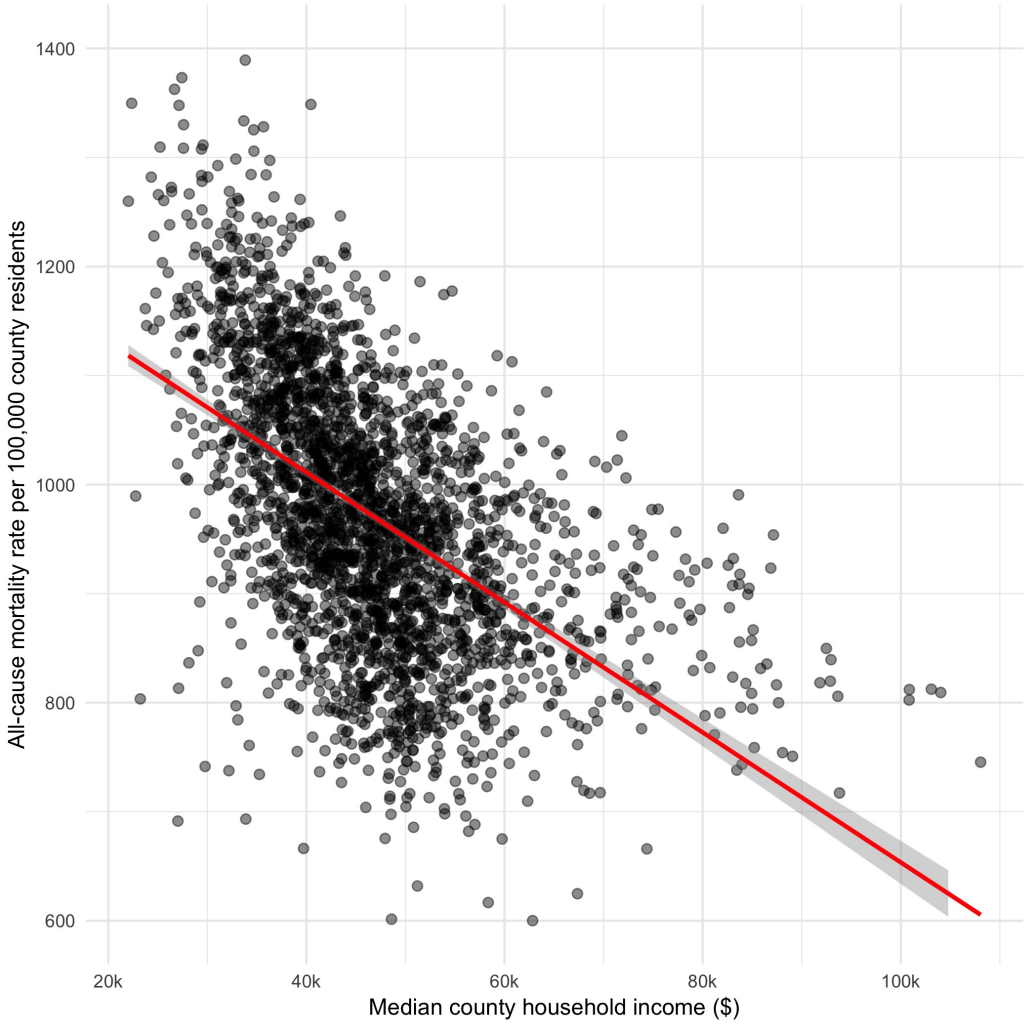

Figure 5.4: *Bivariate association between median household income and age-standardised all-cause mortality (1980–2014 average).*

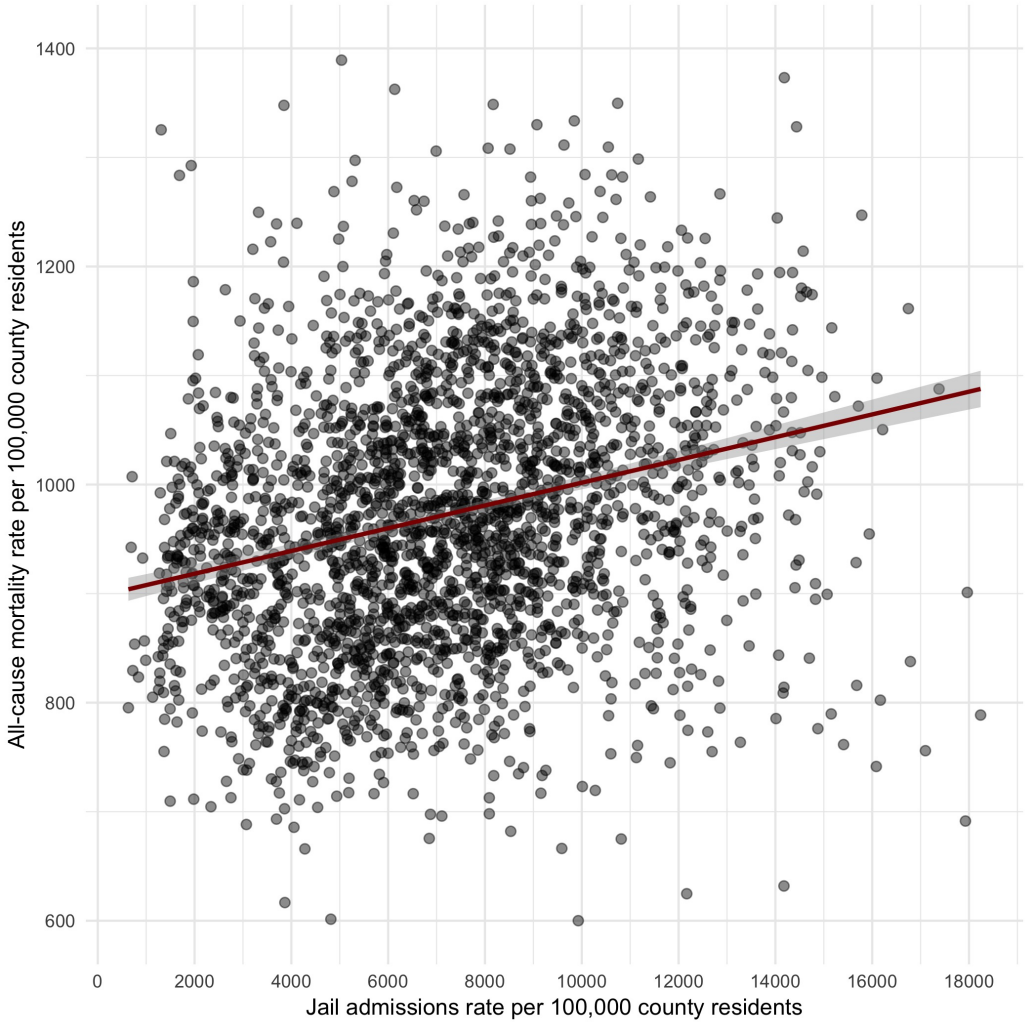

Figure 5.5: *Bivariate association between jail admissions rate and age-standardised all-cause mortality (1980–2014 average).*

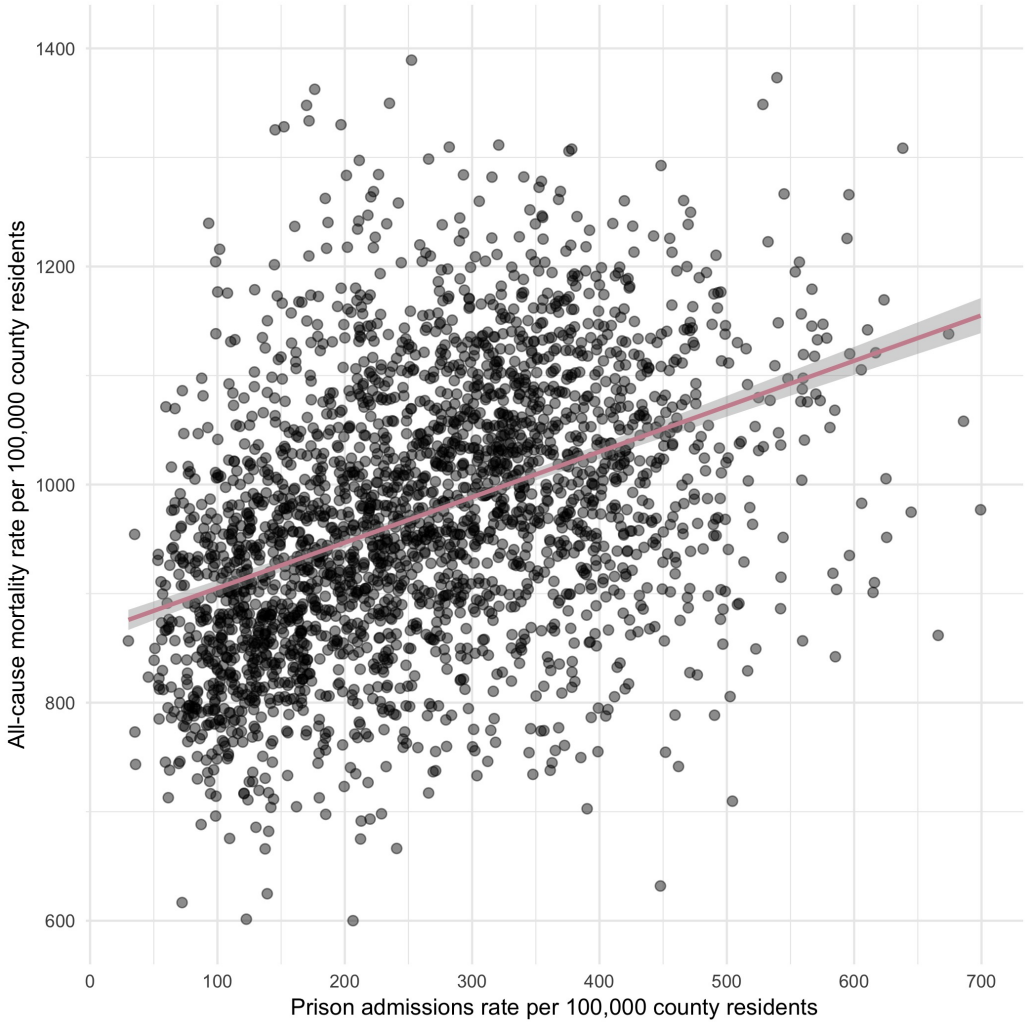

Figure 5.6: *Bivariate association between prison admissions rate and age-standardised all-cause mortality (1980–2014 average).*

posed in chapter 3. Once again, some of the other coefficients are difficult to interpret, and must be read with caution. For instance, in this model, a higher fraction of high school graduates is associated with a higher mortality rate ($\beta = 6.5\%$; 95% CI: [6.0, 7.0]; $P < 0.001$). Given that such variables are nearly time-invariant, the corresponding coefficients may be considered unreliable.

I assess whether a significant portion of the variation in mortality lies between rather than within counties. The variance partition coefficient (VPC) is calculated from a multilevel "null" model and is equal to 74.2%. This means that around three-quarters of the total variation in all-cause mortality lies *between* counties, pointing once again to the utility of a "within-between" specification. The baseline "within-between" model without any control variables (apart from year dummies) is displayed in Table 5.4. The within-county estimates are similar to those of the fixed effects regression: for each one standard deviation increase in jail and prison admissions rates, all-cause mortality rates are associated with a 0.7% (95% CI: [0.6, 0.8]; $P < 0.001$) and a 1.5% (95% CI: [1.4, 1.6]; $P < 0.001$) increase, respectively. Between counties, a one standard deviation rise in jail and prison admissions rates is associated with a 1.2% (95% CI: [0.7, 1.7]; $P < 0.001$) and a 5.4% (95% CI: [4.9, 5.8]; $P < 0.001$) increase, respectively. Comparing the baseline model to the "null" model reveals that the addition of both incarceration predictors reduces the intercept (between-county) variance by over 25% and the residual (within-county) variance by nearly 80%. Figure 5.7 visualises the parameter estimates and corresponding confidence intervals for each of the control models. The reader will note that the "between" estimates for jail

Table 5.3: Fixed effects panel regression model of all-cause mortality rates

	Coefficient	Standard error	P-value
Jail admissions rate	0.5%	0.1	< 0.001
Prison admissions rate	1.2%	0.1	< 0.001
Household income decline	3.2%	0.2	< 0.001
Fraction high school graduates	6.5%	0.3	< 0.001
Fraction African Americans	2.8%	0.7	< 0.001
Fraction Hispanics	1.7%	0.4	< 0.001
Fraction other ethnicity	-3.0%	0.6	< 0.001
Violent crime rate	0.5%	0.1	< 0.001

Notes: The outcome variable is the natural logarithm of the age-standardised county all-cause mortality rate; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model controls for decline in median county household income, the county fraction of high school graduates, African Americans, Hispanics, or other non-White ethnicity, the county violent crime rate, and for aggregate annual time trends using year dummies (not displayed); robust panel-corrected standard errors are presented in the second column; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the all-cause mortality rate associated with a one standard deviation increase in each predictor. $N = 57,732$. $R^2 = 85.6\%$.

admissions rates is rendered statistically insignificant when controlling for reduced median household income or higher high school graduation rates. The "within" estimates remain robust to such controls, however, as do the coefficients for prison admissions rates across all models. Furthermore, the "between" coefficient for education has reversed sign, as expected, meaning the larger the fraction of high school graduates, the lower the all-cause mortality rate. As for the ethno-racial composition of counties, the model indicates a persistent mortality disadvantage for African Americans, but an advantage for Hispanics (consistent with the so-called "Hispanic health paradox"), whereas other ethnic minorities are more ambiguously positioned. As expected, higher rates of violent crime are associated with higher mortality rates.

The matching algorithm generates a pruned data set with $N = 963$. As in the previous chapter, I first estimate the expected value of the outcome variable for a county that is in the "control" group, meaning it has an incarceration rate at one standard deviation below the global mean. As seen in the first density plot (in gray) in Figure 5.8, its age-standardised all-cause mortality rate is equal to 901.0 deaths per 100,000 county residents (95% CI: [892.4, 909.6]; $P < 0.001$). Next, I estimate the expected value of the all-cause mortality rate for a county that only differs from the first in that it has incarceration rates that lie at one standard deviation above the global mean. As evidenced by the second (black) density plot, this raises the mortality rate from 901.0 to 955.8 deaths per 100,000 county residents (95% CI: [947.1, 964.5]; $P < 0.001$). As conveyed by the third and final density (in red), which shows the first difference in the expected value of the mortality

Table 5.4: "Within-between" panel regression model of all-cause mortality

		Coefficient	Standard error	P-value
Jail admissions rate				
	Within	0.7%	0.02	< 0.001
	Between	1.2%	0.2	< 0.001
Prison admissions rate				
	Within	1.5%	0.02	< 0.001
	Between	5.4%	0.2	< 0.001

Notes: The outcome variable is the natural logarithm of the age-standardised county all-cause mortality rate; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model only controls for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the all-cause mortality rate associated with a one standard deviation increase in each predictor. $N = 64,814$.

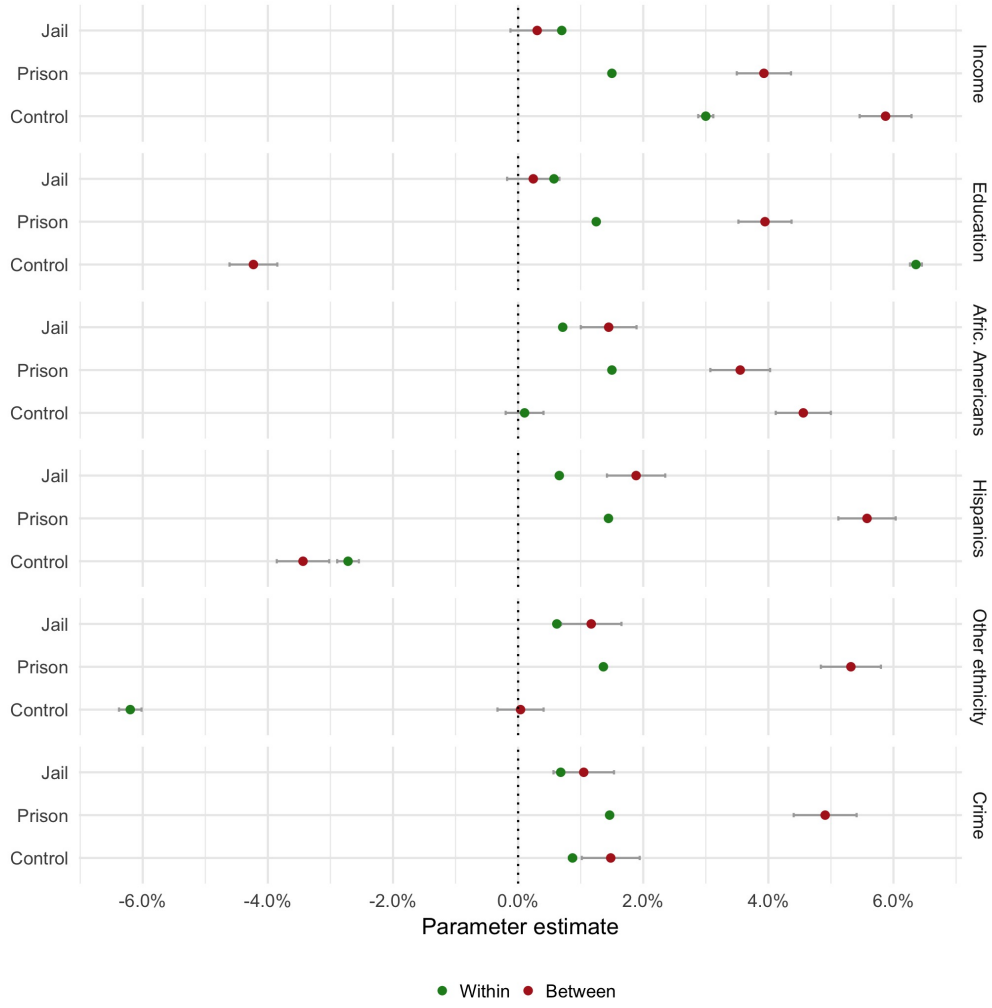

Figure 5.7: *Coefficient plot of multilevel "within-between" random effects models. The outcome variable is the natural logarithm of the age-standardised county all-cause mortality rate; the main predictors are the county jail and prison admissions rates per 100,000 population, adjusted for aggregate annual time trends using year dummies; control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the all-cause mortality rate associated with a one standard deviation increase in each predictor.*

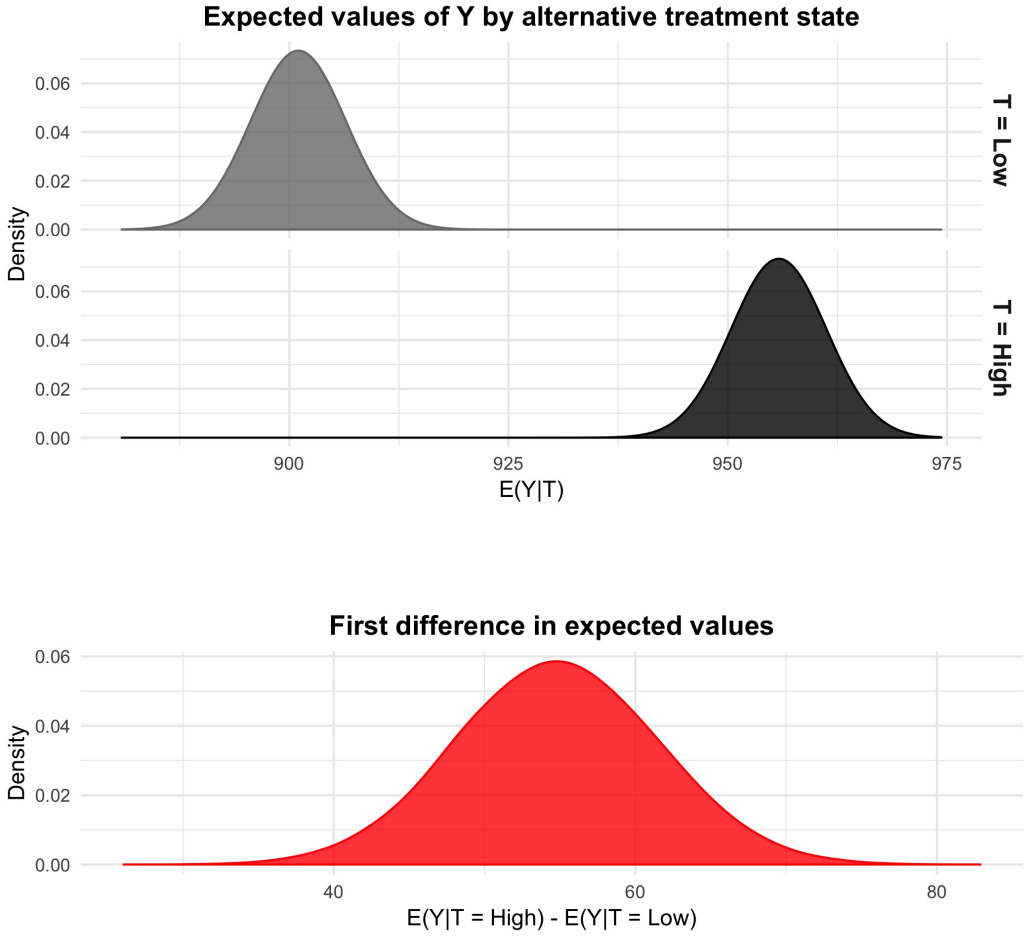

Figure 5.8: *Density plots of simulated expected values of the all-cause mortality rate given a "treatment" (T) state. The outcome variable is the age-standardised all-cause mortality rate; the model compares counties with incarceration rates at one standard deviation below the global mean ($T = 0$) to those with incarceration rates at one standard deviation above the global mean ($T = 1$); the association between all-cause mortality and "treatment" is estimated by applying a simple linear regression model to a pruned data set that is pre-processed using coarsened exact matching; counties are matched on the same variables as in Figure 4.9 in the previous chapter. $N = 963$.*

rate for the different treatment states, these results suggest that, on average, the experience of high incarceration rates corresponds to 54.8 excess deaths per 100,000 county residents (95% CI: [41.7, 68.0]; $P < 0.001$). In terms of the semi-elasticity of Y with respect to T, this result suggests a "treatment effect" equal to a 6.1% increase in the all-cause death rate.

5.3.2 Premature mortality risk

Figure 5.9 shows a strong bivariate association between median household income and the probability of death between the ages of 25 and 45, even more so than for the all-cause mortality in Figure 5.4. Figures 5.10 and 5.11 highlight once again that incarceration may play a central role in the making of this association.

Table 5.5 shows the results of the fixed effects regression model of premature mortality risk. The coefficients are very similar to those displayed for all-cause mortality rates in Table 5.3. For instance, for each standard deviation increase in jail or prison admissions rates, the premature mortality risk is expected to increase by 0.5% (95% CI: [0.4, 0.6]; $P < 0.001$) and 1.2% (95% CI: [1.1, 1.3]; $P < 0.001$), respectively. However, in this case, the VPC is equal to 91.5%, meaning the vast majority of the total variation in premature mortality risk is located between rather than within counties.

The baseline "within-between" model without controls is shown in Table 5.6 and the control models are shown in Figure 5.12. In these models, both kinds of incarceration are robust to all controls and, once again, the "between" estimates provide more meaningful results for some of the other covariates, especially education. As expected, the "between" effects tend to

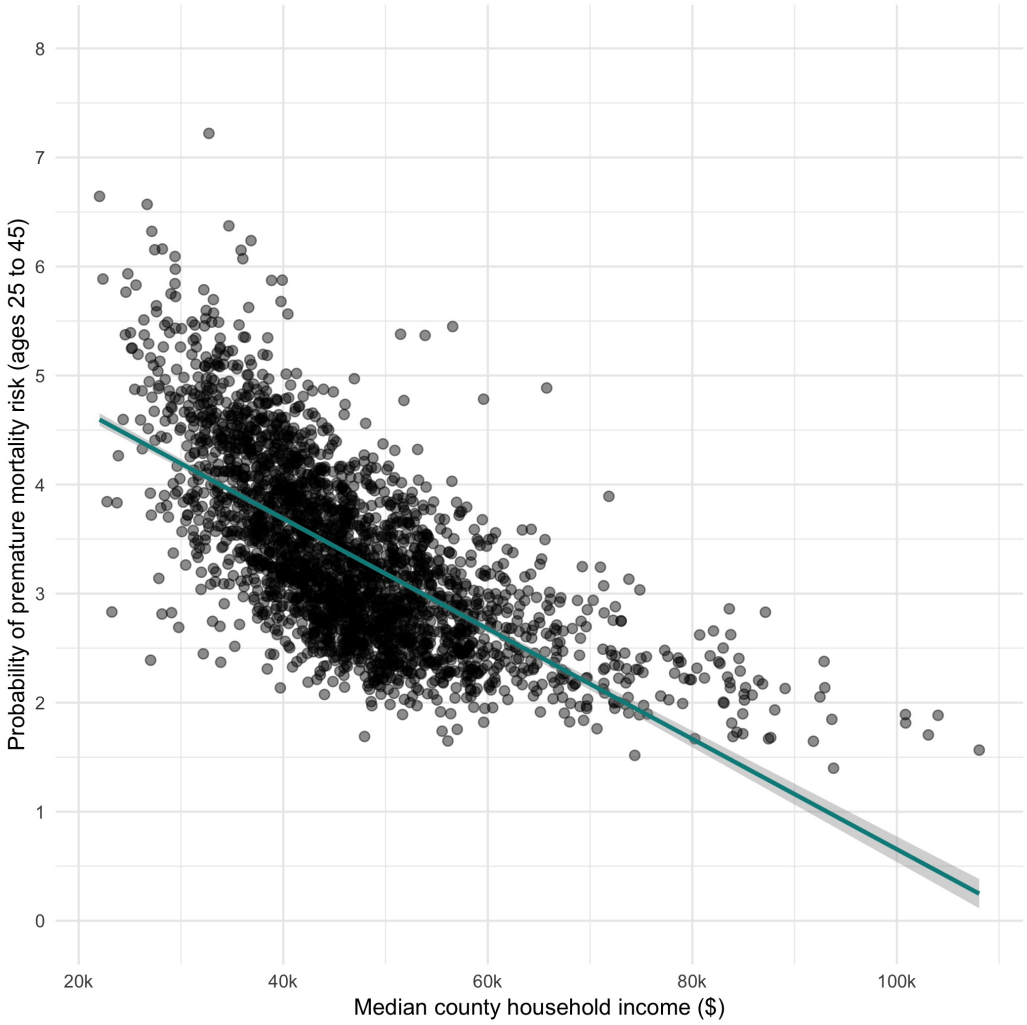

Figure 5.9: *Bivariate association between median household income and probability of premature death (1980–2014 average).*

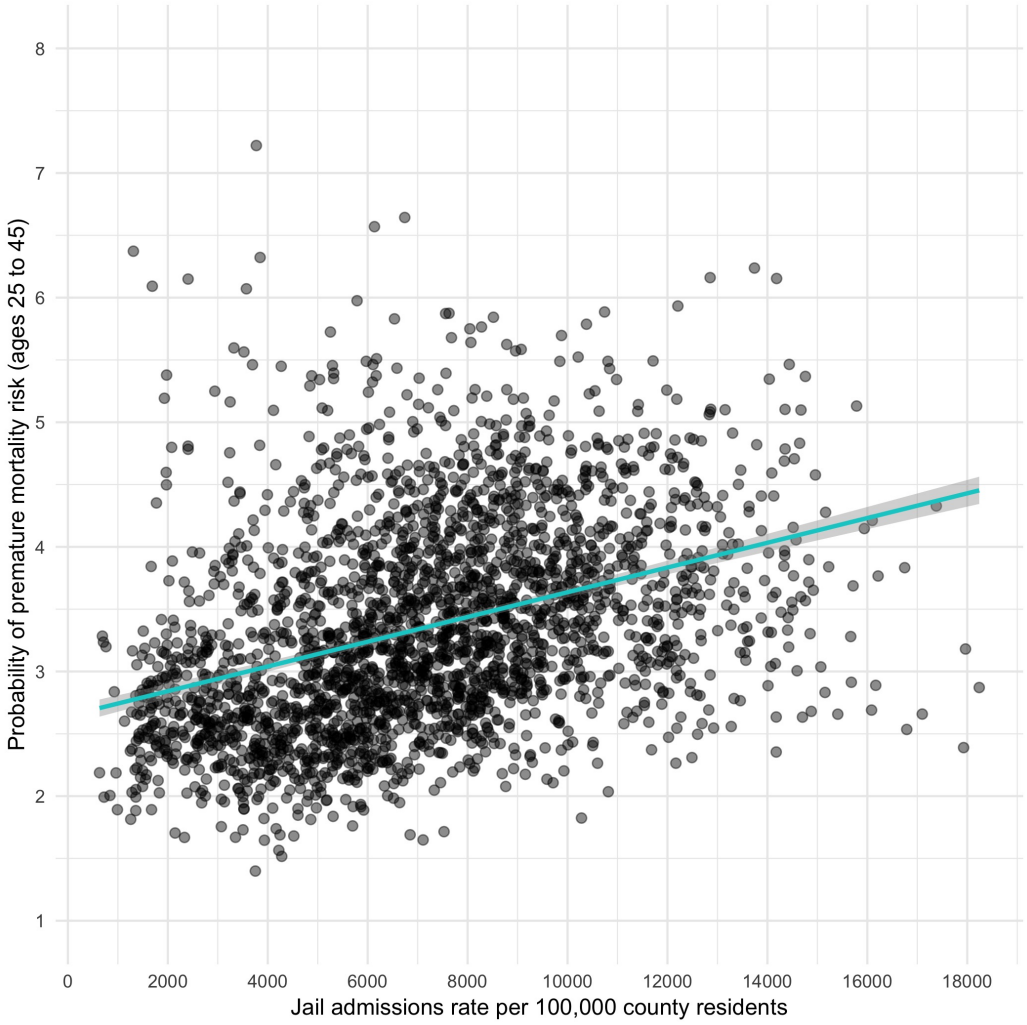

Figure 5.10: *Bivariate association between jail admissions rate and probability of premature death (1980–2014 average).*

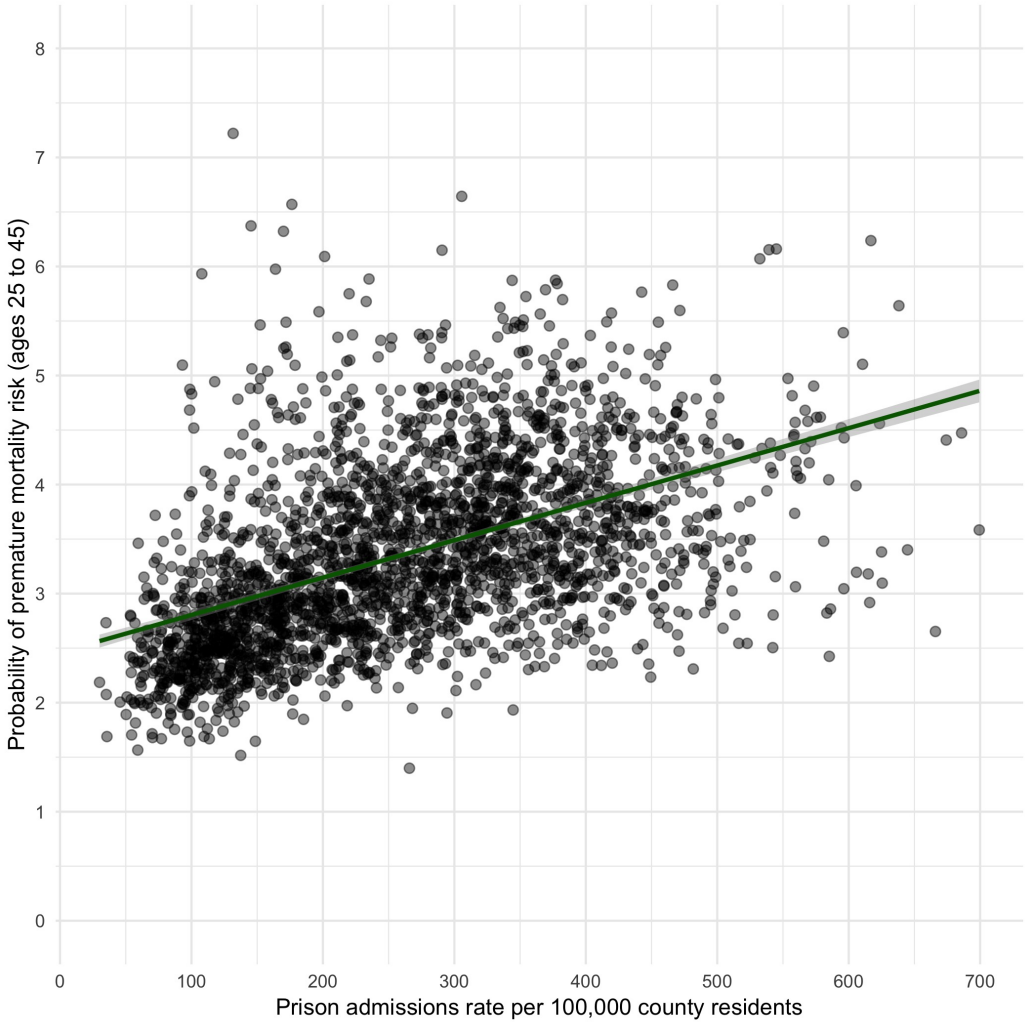

Figure 5.11: *Bivariate association between prison admissions rate and probability of premature death (1980–2014 average).*

Table 5.5: Fixed effects panel regression model of premature mortality risk

	Coefficient	Standard error	P-value
County jail admissions rate	0.5%	0.1	< 0.001
County prison admissions rate	1.2%	0.1	< 0.001
Household income decline	3.3%	0.2	< 0.001
Fraction high school graduates	6.9%	0.3	< 0.001
Fraction African Americans	4.4%	0.9	< 0.001
Fraction Hispanics	1.7%	0.4	< 0.001
Fraction other ethnicity	-3.2%	0.6	< 0.001
Violent crime rate	0.5%	0.1	< 0.001

Notes: The outcome variable is the natural logarithm of the premature mortality risk between the ages of 25 and 45; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model controls for decline in median county household income, the county fraction of high school graduates, African Americans, Hispanics, or other non-White ethnicity, the county violent crime rate, and for aggregate annual time trends using year dummies (not displayed); robust panel-corrected standard errors are presented in the second column; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the premature mortality risk associated with a one standard deviation increase in each predictor. $N = 57,732$. $R^2 = 82.0\%$.

Table 5.6: "Within-between" panel regression model of premature mortality risk

		Coefficient	Standard error	P-value
Jail admissions rate				
	Within	0.7%	0.03	< 0.001
	Between	4.7%	0.4	< 0.001
Prison admissions rate				
	Within	1.5%	0.02	< 0.001
	Between	11.1%	0.4	< 0.001

Notes: The outcome variable is the natural logarithm of the premature mortality risk between the ages of 25 and 45; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model only controls for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the premature mortality risk associated with a one standard deviation increase in each predictor. $N = 64,814$.

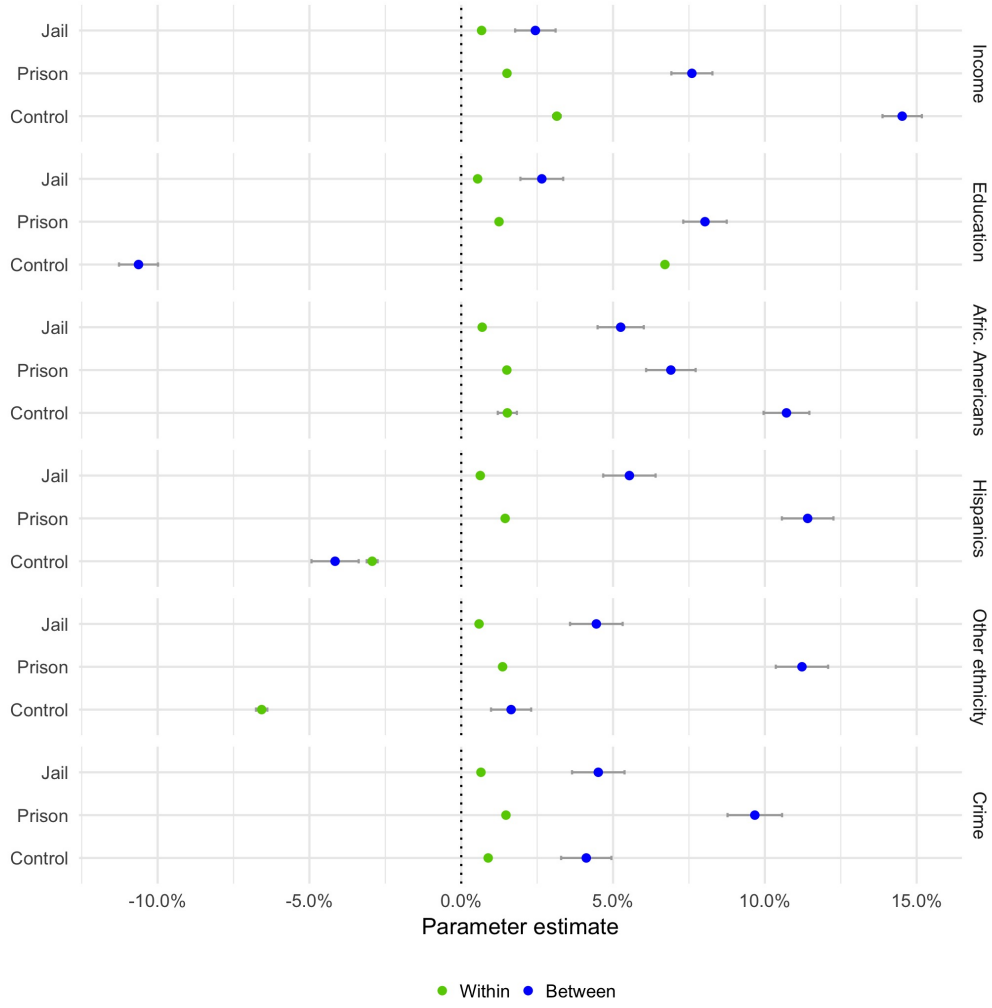

Figure 5.12: *Coefficient plot of multilevel "within-between" random effects models. The outcome variable is the natural logarithm of the premature mortality risk between the ages of 25 and 45; the main predictors are the county jail and prison admissions rates per 100,000 population, adjusted for aggregate annual time trends using year dummies; control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the percentage change in the premature mortality risk associated with a one standard deviation increase in each predictor.*

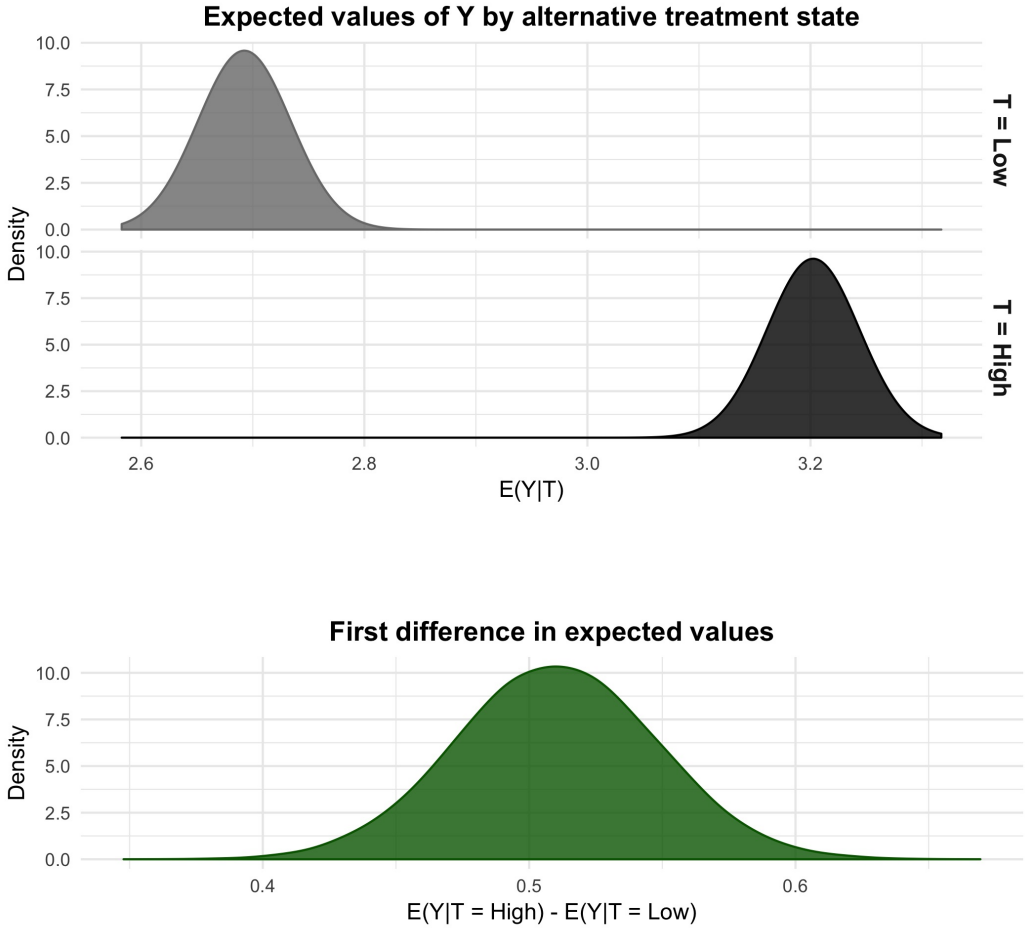

Figure 5.13: *Density plots of simulated expected values of premature mortality risk given a continuous "treatment" (T) state. The outcome variable is the premature mortality risk; the model compares counties with incarceration rates at one standard deviation below the global mean ($T = 0$) to those with incarceration rates at one standard deviation above the global mean ($T = 1$); the association between premature mortality risk and "treatment" is estimated by applying a simple linear regression model to a pruned data set that is pre-processed using coarsened exact matching; counties are matched on the same variables as in Figure 4.9 in the previous chapter. $N = 963$.*

be larger than the "within" effects. In the baseline model, each standard deviation increase in jail or prison rates is associated with a 0.7% (95% CI: [0.6, 0.7]; $P < 0.001$) and a 1.5% (95% CI: [1.4, 1.6]; $P < 0.001$) increase in the premature mortality risk within counties, respectively. Between counties, the corresponding figures are 4.7% (95% CI: [3.9, 5.6]; $P < 0.001$) and 11.1% (95% CI: [10.3, 12.0]; $P < 0.001$), respectively. Comparing the baseline model to the "null" model shows that the addition of both incarceration predictors reduces the intercept ("between") variance by a remarkable 94% and the residual ("within") variance by 98%.

The results of the simulation-based "between" model are shows in Figure 5.13. Counties in the "control" group have an average premature mortality risk equal to 2.7% (95% CI: [2.6, 2.7]; $P < 0.001$), whereas counties with high incarceration rates have an average premature mortality risk equal to 3.2% (95% CI: [3.1, 3.2]; $P < 0.001$). The first difference is half a percentage point (95% CI: [0.4, 0.6]; $P < 0.001$), which amounts to a semi-elasticity of 19.0% in the probability of death between the ages of 25 and 45 with respect to high rates of incarceration.

5.3.3 Life expectancy

Figures 5.14, 5.15, and 5.16 visualise the same bivariate associations as were presented above for all-cause mortality rates and premature mortality risk, only this time for life expectancy at birth. As expected, there seems to be a strong relationship between economic deprivation and life expectancy, but also between incarceration rates and life expectancy.

The results for the fixed effects regression are shown in Table 5.7. Here

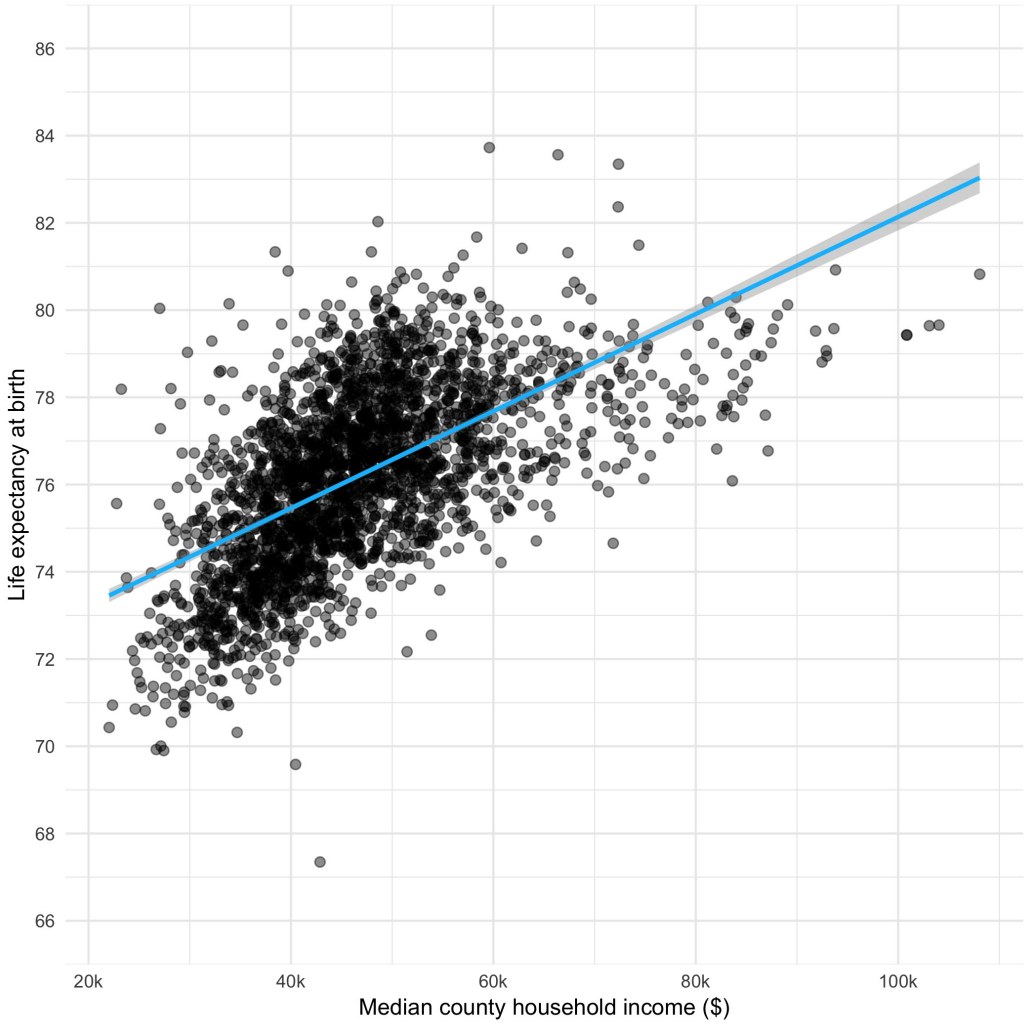

Figure 5.14: *Bivariate association between median household income and life expectancy at birth (1980–2014 average).*

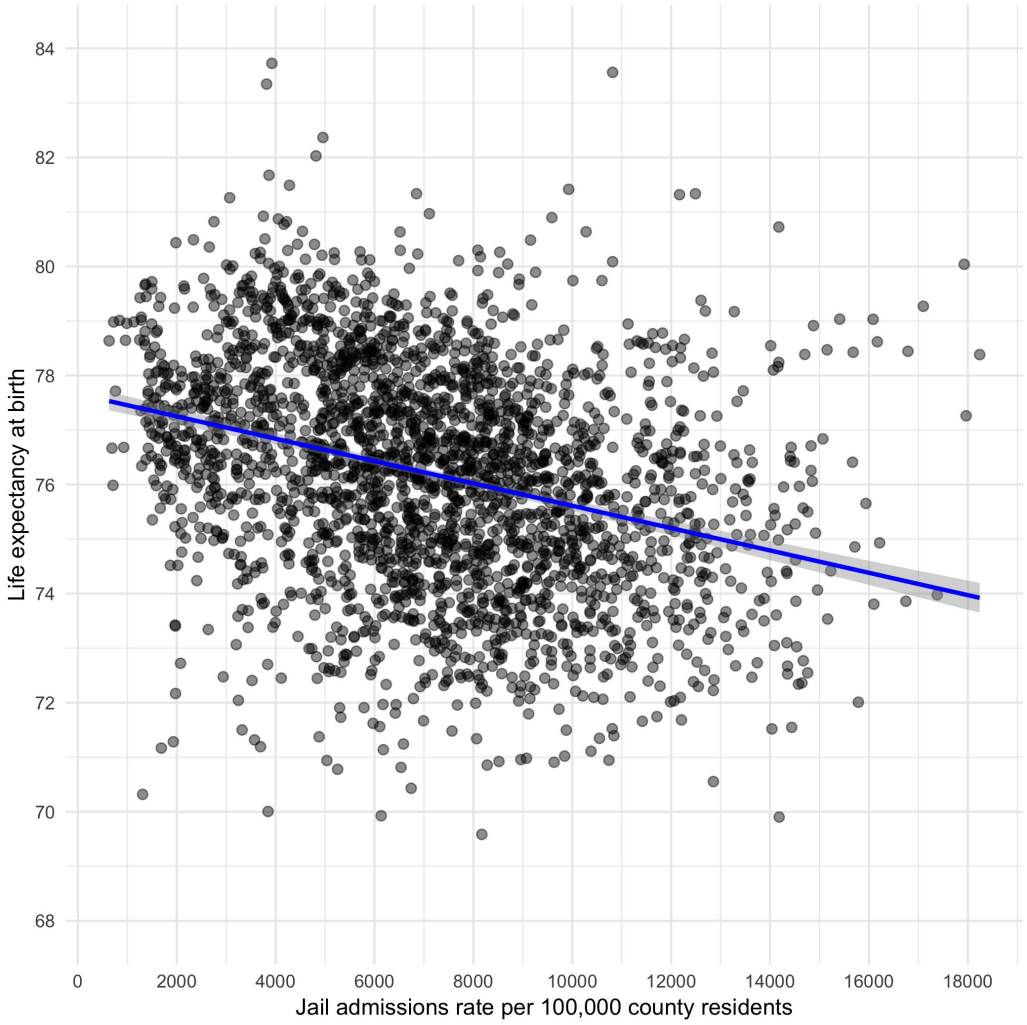

Figure 5.15: *Bivariate association between jail admissions rate and life expectancy at birth (1980–2014 average).*

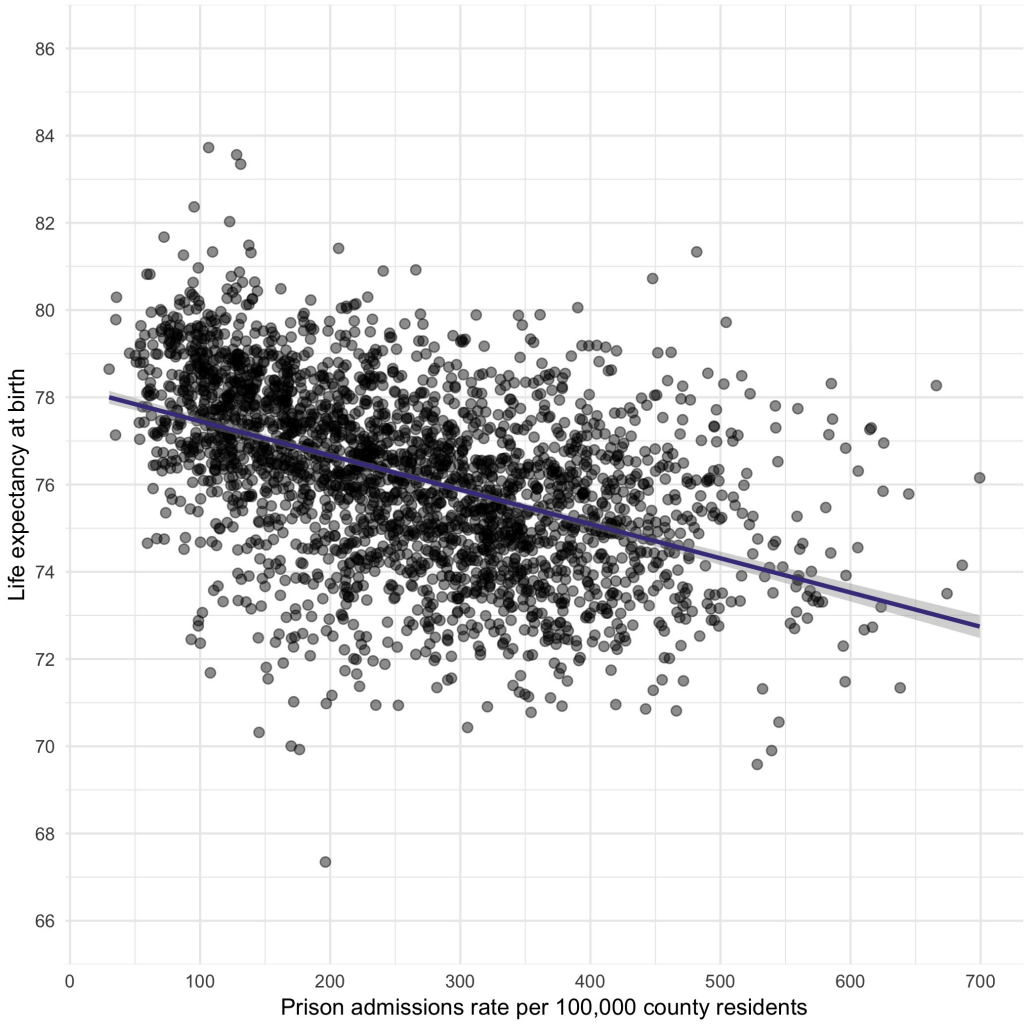

Figure 5.16: *Bivariate association between prison admissions rate and life expectancy at birth (1980–2014 average).*

the outcome variable is not log-transformed, thus rendering coefficients interpretable as the change (in years) in life expectancy associated with a one standard deviation increase in each regressor. Thus longevity at the county level is expected to decrease by 0.06 years (95% CI: [0.045, 0.073]; $P < 0.001$) for each rise in jail admissions rates and by over one-tenth of a year (β = -0.14; 95% CI: [-0.15, -0.12]; $P < 0.001$) for each increase in prison admissions rates. Reduced median household income is associated with a four-month decline in life expectancy (β = -0.32; 95% CI: [-0.37, -0.27]; $P < 0.001$), whilst violent crime has a similar coefficient to jail incarceration (β = -0.06; 95% CI: [-0.08, -0.04]; $P < 0.001$). As noted earlier, the other coefficients are more readily interpretable in an alternative model specification. Also in this model, a substantial amount of variation in life expectancy is captured ($R^2 = 91.4\%$).

A multilevel "null" model reveals a VPC equal to 77.2%, meaning over three-quarters of the total variation in life expectancy at birth lies between rather than within counties. The baseline "within-between" model is shown in Table 5.8. The "within" estimates are largely similar to those produced by the fixed effects regression. The "between" estimates suggest that for each standard deviation increase in incarceration rates, life expectancy is expected to decrease by about a quarter of a year in the case of jails (β = -0.26; 95% CI: [-0.33, -0.20]; $P < 0.001$) and by almost a full year in the case of prisons (β = -0.90; 95% CI: [-1.00, -0.83]; $P < 0.001$). Comparing the baseline model to the "null" model reveals that the addition of both incarceration predictors reduces the intercept ("between") variance by over 30% and the residual ("within") variance by 88%.

Table 5.7: Fixed effects panel regression model of life expectancy at birth

	Coefficient	Standard error	P-value
County jail admissions rate	-0.06	0.01	< 0.001
County prison admissions rate	-0.14	0.01	< 0.001
Household income decline	-0.32	0.02	< 0.001
Fraction high school graduates	-0.60	0.03	< 0.001
Fraction African Americans	-0.38	0.09	< 0.001
Fraction Hispanics	-0.11	0.05	0.03
Fraction other ethnicity	0.29	0.07	< 0.001
Violent crime rate	-0.06	0.01	< 0.001

Notes: The outcome variable is county-level life expectancy at birth; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model controls for decline in median county household income, the county fraction of high school graduates, African Americans, Hispanics, or other non-White ethnicity, the county violent crime rate, and for aggregate annual time trends using year dummies (not displayed); robust panel-corrected standard errors are presented in the second column; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 57,732$. $R^2 = 91.4\%$.

Table 5.8: "Within-between" panel regression of life expectancy at birth

		Coefficient	Standard error	P-value
Jail admissions rate				
	Within	-0.08	0.003	< 0.001
	Between	-0.26	0.04	< 0.001
Prison admissions rate				
	Within	-0.17	0.003	< 0.001
	Between	-0.90	0.04	< 0.001

Notes: The outcome variable is county-level life expectancy at birth; the main predictors are the county jail admissions rate and the county prison admissions rate per 100,000 population; the model only controls for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 64,814$.

The control models are visualised in Figure 5.17, which suggests that the above estimates are robust. Both jail and prison admissions rates are significantly associated with reduced life expectancy in all models. For all regressors, the "within" and "between" estimates carry the same sign, with the exception of high school graduation rates – which, as expected, are associated with *higher* life expectancy between counties – and non-White ethnic minorities other than African Americans and Hispanics, whose residential concentration is associated with lower life expectancy. Otherwise, also African Americans suffer from reduced longevity, whereas Hispanics tend to live longer. As expected, low income or income decline and violent crime are associated with lower life expectancy.

Figure 5.18 shows the results of the simulation-based modelling approach. Counties with low incarceration rates have an average life expectancy of 77.6 years (95% CI: [77.4, 77.7]; $P < 0.001$), whereas counties with high incarceration rates have an average life expectancy of 76.5 years (95% CI: [76.4, 76.6]; $P < 0.001$). The first difference in expected values is -1.1 years (95% CI: [-1.3, -0.9]; $P < 0.001$). In other words, high rates of incarceration are associated with life expectancy at birth being reduced by over a whole year.

5.4 Discussion

The findings of this chapter seem to confirm the hypothesis that high rates of incarceration can shape the dynamics of vital inequality beyond its effects on the overdose epidemic. However, the same limitations as were highlighted

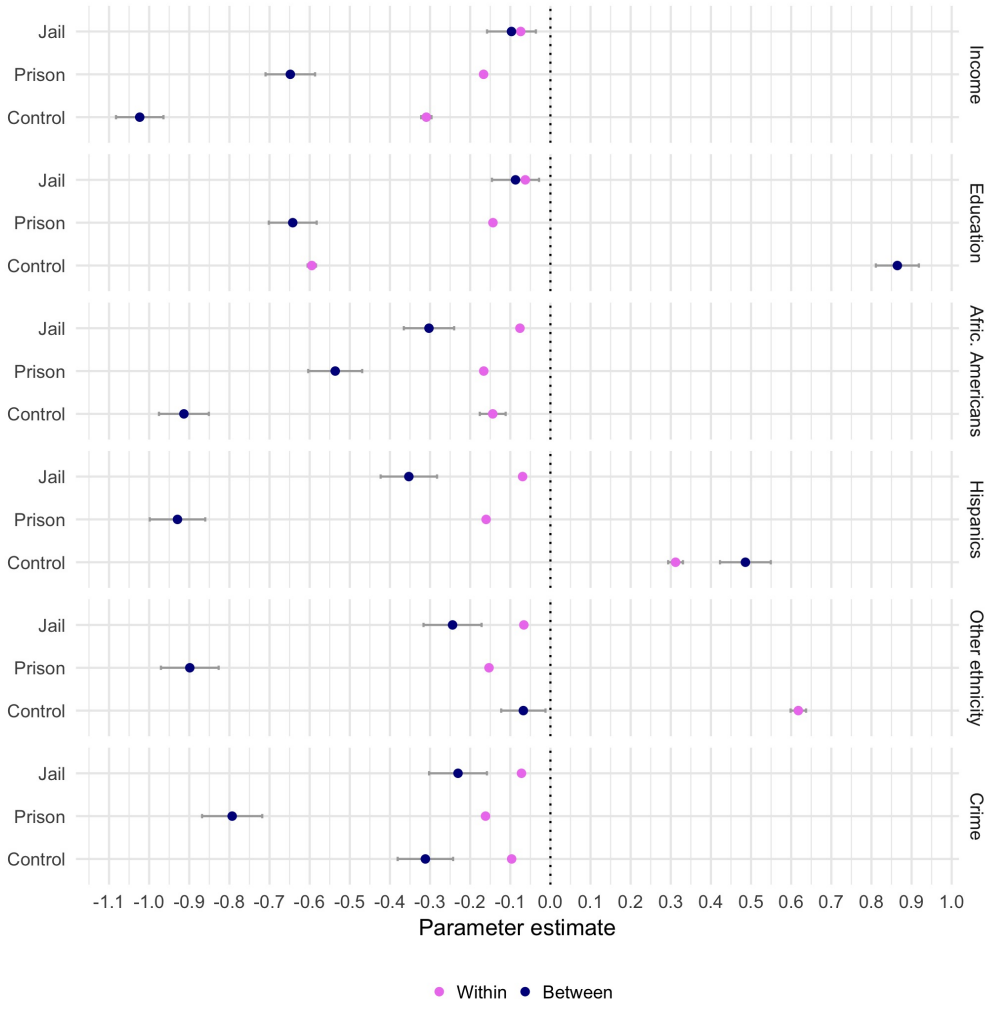

Figure 5.17: *Coefficient plot of multilevel "within-between" random effects models. The outcome variable is county-level life expectancy at birth; the main predictors are the county jail and prison admissions rates per 100,000 population, adjusted for aggregate annual time trends using year dummies; control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor.*

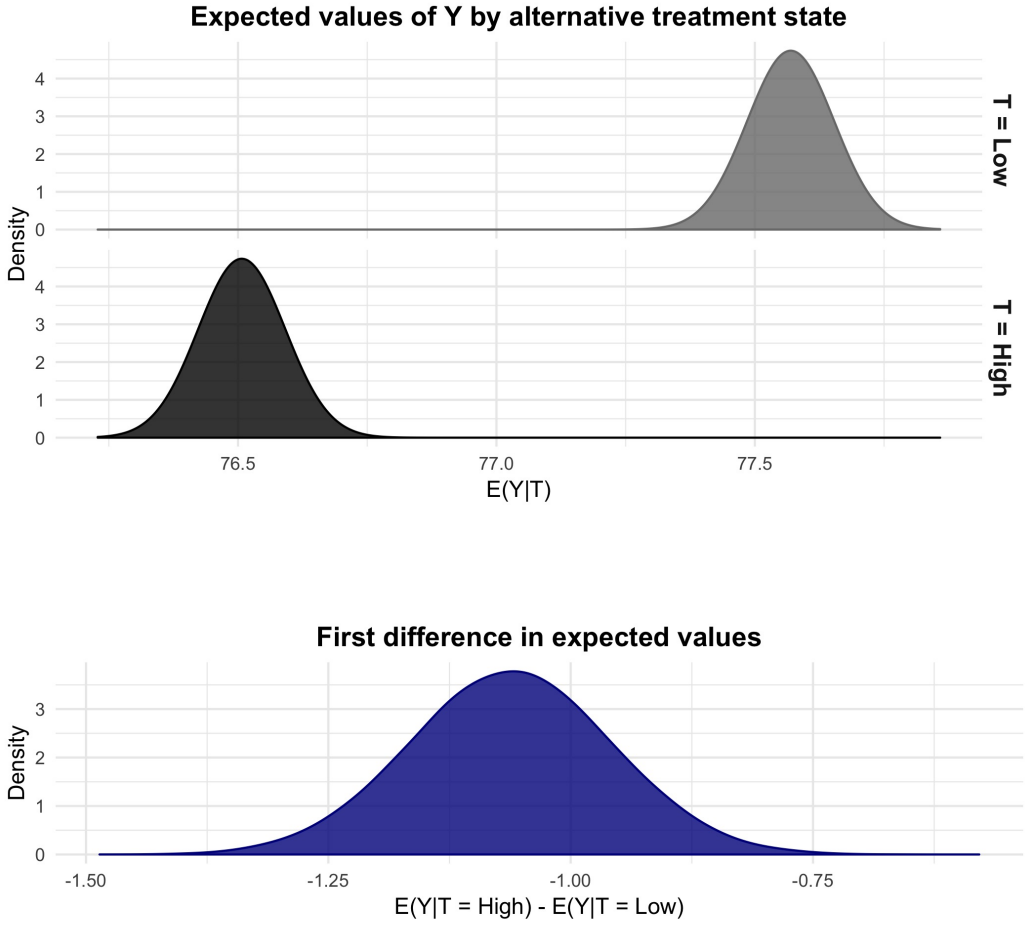

Figure 5.18: *Density plots of simulated expected values of life expectancy at birth given a continuous "treatment" (T) state. The outcome variable is the premature mortality risk; the model compares counties with incarceration rates at one standard deviation below the global mean ($T = 0$) to those with incarceration rates at one standard deviation above the global mean ($T = 1$); the association between life expectancy and "treatment" is estimated by applying a simple linear regression model to a pruned data set that is pre-processed using coarsened exact matching; counties are matched on the same variables as in Figure 4.9 in the previous chapter. $N = 963$.*

in the previous chapter also apply here. Most notably, the data limitations and research design preclude any strict causal inference to be drawn from the analysis although jail and prison incarceration rates seem to be deeply implicated in deepening inequalities in health and wellbeing in the United States. Moreover, although I have hypothesised about a number of plausible mechanisms by which penal expansion may affect the health of individuals, local communities, and even entire populations, the challenge remains to empirically flesh these out. However, as noted before, this would require access to high-quality multilevel data.

That being said, the above analyses suggest a robust association between the rise of the penal state and vital inequality, as measured in three different ways, namely all-cause mortality, premature mortality risk, and life expectancy at birth. Especially for inequalities in premature mortality risk, incarceration accounts for an astounding proportion of variation both within and between counties. This association holds up across a number of different model specifications and provides reasonable estimates of the magnitude of the relevant relationships. Once again, light was shed on the role of a hitherto largely neglected component of the American criminal justice system, namely jail incarceration, and a substantial amount of variation within and between counties is accounted for, especially when it comes to premature mortality risk.

5.5 Conclusion

Both jails and prisons may be construed as vectors of vital inequality in the United States. This chapter shows that high rates of incarceration seem to affect population health not only through rising mortality from drug use disorders, as was documented in the previous chapter, but also through other pathways that shape the social and spatial patterning of disability, disease, and death. The findings suggest that in the wake of economic decline and deepening social division, protective rather than punitive political responses may help to shield vulnerable populations from attendant deleterious health consequences in the form of all-cause mortality, premature death, and life expectancy.

Chapter 6

The association between income and health revisited

6.1 Introduction

In a recent paper, Raj Chetty and colleagues (2016) examine the relationship between income and life expectancy in the United States between 2001 and 2014. They demonstrate how life expectancy tends to rise with income and how health inequalities between top and bottom income groups have widened rapidly over time. Moreover, whilst the rich tend to live longer everywhere, life expectancy amongst the poor shows significant geographical variation. The authors suggest a role for local area characteristics but refrain from further analysis. In this chapter, I seek to examine state-level determinants of life expectancy in the bottom income quartile. At the cost of lower geographical (and temporal) resolution than in previous chapters, I expand the investigation of economic decline, which thus far has only been

operationalised as reduced median household income. In the present chapter, I study variation in longevity amongst those in the bottom quartile of the American income distribution, with an eye on how such variation may be shaped by a different economic factor, namely the fragmentation of labour through deindustrialisation, as was described in chapter 2.

6.2 Empirical strategy

Data

My principal outcome variable is state-level life expectancy at age 40 in the bottom quartile of the income distribution between 2001 and 2014 for all 50 states. These public-use data from the Health Inequality Project (2016) are generated from 1.4 billion tax records between 1999 and 2014 linked to mortality data from Social Security Administration death records. Deindustrialisation is measured by the annual state-level job destruction rate in the manufacturing sector (North American Industry Classification System sector 31-33), i.e. the number of jobs lost to establishment contraction or closure in a year divided by the employment at the beginning of the year. Data on employment and job destruction come from the U.S. Census Statistics of U.S. Businesses Employment Change Data Tables. State-level incarceration rates from the Bureau of Justice Statistics express the count of prisoners serving sentences of more than 1 year per 100,000 state residents. These are prison – not jail – incarceration rates, since prisons are state-level institutions whereas jails are run by local counties within states.

I draw on a series of data sources to conduct a sensitivity analysis. I

calculate the state fraction of those earning less than $25,000 per annum (p.a.) who are overweight or obese; who, at the time of being surveyed, have gone without physical exercise in the past 30 days; who are current smokers; and who are without any form of health insurance. The same variables are also calculated for those earning more than $75,000 p.a. as proxy controls for the top income quartile. These income thresholds, roughly corresponding to the top and bottom income quartiles, are the ones defined by the Centers for Disease Control and Prevention's survey design. I also assess the robustness of the main predictors to state government expenditure on workers' insurance, healthcare, and welfare, as well as the state drug overdose and homicide rates, state GDP per capita, economic growth, the relative size of the manufacturing sector, and labour force participation rates. Variable definitions and sources are listed below, descriptive statistics are presented in Table 6.1, and a Spearman correlation matrix is presented in Table 6.2.

- Social spending. *Definition*: amount (in U.S. dollars) spent by state government in each fiscal year on workers' insurance trusts divided by state population. *Source*: U.S. Census Bureau: State Government Finances.

- Health spending. *Definition*: amount (in U.S. dollars) spent by state government in each fiscal year on healthcare divided by state population. *Source*: U.S. Census Bureau: State Government Finances.

- Welfare spending. *Definition*: amount (in U.S. dollars) spent by state government in each fiscal year on public welfare divided by state pop-

ulation. *Source*: U.S. Census Bureau: State Government Finances.

- Fraction obese. *Definition*: fraction of individuals earning less than $25,000 p.a./more than $75,000 p.a. who are either overweight or obese. *Source*: Centers for Disease Control and Prevention: Behavioral Risk Factor Surveillance System.

- Fraction physically inactive. *Definition*: fraction of individuals earning less than $25,000 p.a./more than $75,000 p.a. who have not engaged in physical exercise in the past 30 days. *Source*: Centers for Disease Control and Prevention: Behavioral Risk Factor Surveillance System.

- Fraction smokers. *Definition*: fraction of individuals earning less than $25,000 p.a./more than $75,000 p.a. who are current smokers. *Source*: Centers for Disease Control and Prevention: Behavioral Risk Factor Surveillance System.

- Fraction uninsured. *Definition*: fraction of individuals earning less than $25,000 p.a./more than $75,000 p.a. without any form of medical insurance. *Source*: Centers for Disease Control and Prevention: Behavioral Risk Factor Surveillance System.

- Overdose mortality rate. *Definition*: number of state level deaths per 100,000 state residents amongst individuals aged 20-64 years. *Source*: Centers for Disease Control and Prevention: Compressed Mortality database (codes X40-44, X60-64, X85, Y10-14).

- Homicide rate. *Definition*: total number of murders committed per 100 000 state residents. *Source*: Federal Bureau of Investigation: Uniform

Crime Reporting Statistics.

- GDP per capita. *Definition*: state real gross domestic product in thousands of U.S. dollars divided by state population estimate. *Source*: Bureau of Economic Analysis: Regional Economic Accounts.

- GDP growth. *Definition*: annual change in state real gross domestic product in thousands of U.S. dollars divided by state population estimate. *Source*: Bureau of Economic Analysis: Regional Economic Accounts.

- Size of manufacturing. *Definition*: total state employment in manufacturing sector at the start of each year divided by total employment across all sectors. *Source*: U.S. Census Bureau: Statistics of U.S. Businesses.

- Labour force participation rate. *Definition*: civilian labour force as percentage of total state population. *Source*: Bureau of Labor Statistics: Local Areas Unemployment Statistics.

Methods

Due to the new unit of analysis (the state) and a significantly smaller sample size ($N = 700$) as compared to the previous two chapters, I confine my methodological approach to estimating fixed effects panel regressions. This is because the data lack the statistical power to separately estimate within- and between-state coefficients, and the data pruning effected through matching would result in having virtually no data left for the actual analysis. Instead,

Table 6.1: Descriptive statistics

Statistic	N	Mean	St. Dev.	Min	Max
Life expectancy Q1	700	79.6	1.5	73.9	83.7
Life expectancy Q4	700	87.0	1.6	80.8	91.5
Deindustrialisation	700	11.2	3.5	0.0	27.5
Prison incarceration rate	697	399.7	145.1	127.0	881.0
Social spending	700	695.0	322.9	156.2	1,833
Health spending	700	186.2	98.9	40.8	529.9
Welfare spending	700	1,324	443.6	402.5	2,948
Fraction obese	699	0.6	0.04	0.4	0.7
Fraction physically inactive	694	0.4	0.1	0.2	0.5
Fraction smokers	699	0.3	0.04	0.2	0.4
Fraction uninsured	694	0.2	0.1	0.1	0.4
Overdose mortality rate	700	18.4	7.7	2.6	54.7
Homicide rate	750	4.5	2.3	0.6	14.6
GDP per capita	750	45,804	8,639	28,856	73,464
GDP growth	700	343.5	1,245	−4,512	11,009
Size of manufacturing	650	11.3	4.4	2.4	23.2
Labour force participation rate	700	66.1	4.2	53.3	76.1

Notes: All variables are at the state level between 2001 and 2014; Q1 = bottom income quartile; Q4 = top income quartile; rates are per 100,000 state residents; deindustrialisation is the annual percentage of jobs destroyed in the manufacturing sector; government spending is per capita; both GDP variables are measured in thousands of U.S. dollars.

Table 6.2: Spearman correlation matrix

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.
1. Life expectancy Q1	1																
2. Life expectancy Q4	0.3	1															
3. Deindustrialisation	-0.05	-0.2	1														
4. Incarceration rate	-0.4	-0.1	0.1	1													
5. Social spending	0.2	0.4	-0.2	-0.1	1												
6. Health spending	0.2	0.2	0.02	-0.1	0.3	1											
7. Welfare spending	0.2	0.4	-0.3	-0.3	0.6	0.4	1										
8. Fraction obese	-0.3	0.2	-0.3	0.3	0.2	-0.04	0.4	1									
9. Fraction inactive	-0.5	-0.3	0.2	0.4	-0.2	-0.2	0.01	0.5	1								
10. Fraction smokers	-0.5	-0.3	0.04	0.2	-0.3	-0.1	-0.2	-0.04	0.2	1							
11. Fraction uninsured	-0.002	-0.1	0.1	0.4	-0.1	-0.2	-0.4	-0.2	0.01	0.2	1						
12. Overdose rate	0.03	0.3	-0.1	0.1	0.4	0.2	0.3	0.2	0.1	-0.03	0.1	1					
13. Homicide rate	-0.4	-0.2	0.2	0.7	-0.1	-0.01	-0.1	0.3	0.5	0.2	0.4	0.2	1				
14. GDP per capita	0.2	0.2	-0.01	-0.3	0.4	0.3	0.2	-0.1	-0.1	-0.3	-0.2	-0.1	-0.2	1			
15. GDP growth	0.1	-0.1	-0.3	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	0.1	0.01	-0.2	-0.1	0.1	1		
16. Manufacturing size	-0.5	-0.3	-0.1	0.04	-0.3	-0.4	-0.1	0.2	0.3	0.3	-0.2	-0.3	0.02	-0.4	0.02	1	
17. Labour part. rate	0.2	0.01	-0.01	-0.4	-0.2	-0.1	-0.3	-0.3	-0.3	-0.05	-0.2	-0.4	-0.5	0.4	0.1	0.02	1

I present a baseline fixed effects model adjusted for aggregate time trends, before conducting a sensitivity analysis where state-level control variables are introduced into and removed from the unadjusted baseline model one by one to avoid over-specification. As an additional robustness check, I run alternative control models with multiple control variables grouped into three categories. Given that I am investigating aggregate life expectancy in the bottom income quartile at the state level, I lag the two main predictors by one year to allow for delayed effects to manifest. To assess the degree to which deindustrialisation and incarceration rates not only account for variation in life expectancy amongst the poor, but also drive inequalities between the top and the bottom of the income distribution, I run the same models with life expectancy in the top income quartile as the outcome variable as a point of comparison.

6.3 Findings

Figure 6.1 shows the evolution of the life expectancy gap between the top and bottom income quartiles between 2001 and 2014. The plot suggests a clear increase in inequality over time. In Figure 6.2, life expectancy at age 40 in the bottom income quartile is plotted against job destruction rate in manufacturing, lagged one year, as a measure of deindustrialisation. A linear estimator is used to measure the gradient between the two variables, which is negative. Thus, an increase in deindustrialisation in a given year is negatively associated with life expectancy amongst the poor in the following year. Figure 6.3 is similar, only this time life expectancy at age 40 is plotted

against state level incarceration rates per 100,000 state residents, also lagged one year. The slope is negative and steep, indicating a pronounced inverse association between life expectancy and high imprisonment. The time series plot in Figure 6.4 compares the level of life expectancy in the bottom income quartile between states characterised by low and high incarceration rates over time. The plot conveys how inequalities between low- and high-incarceration states are distinct: poor lives are over one year shorter in states in the top incarceration decile (mean incarceration rate = 694.7 prisoners per 100,000 residents) relative to states in the bottom decile (mean incarceration rate = 185.2 prisoners per 100,000 residents), and there is some indication of a growing gap. Moreover, Figures 6.5 and 6.6 enable an approximate estimation of the long-term effects of deindustrialisation and the legacy of slavery. That former slave states are to incarceration what Rust Belt states are to deindustrialisation is reflected in how eight out of the top ten incarcerator states in this time period are former slave states.

The baseline model is displayed in Table 6.3, indicating that a one standard deviation increase in deindustrialisation (mean = 11.2, standard deviation = 3.5) reduces life expectancy for the poor by 0.26 years (95% CI: [0.10, 0.42]; $P = 0.002$). Relative to the average state, those states characterised by a job destruction rate in manufacturing of 20% or more lost at least another 0.641 years. In the case of incarceration (mean = 399.7, standard deviation = 145.1), each standard deviation increase is associated with a loss of 0.68 years (95% CI: [0.31, 1.05]; $P < 0.001$). Compared to the poor living in the average state, those living in states characterised by high incarceration (such as Louisiana, with a mean incarceration rate of 837.0 prisoners per 100,000

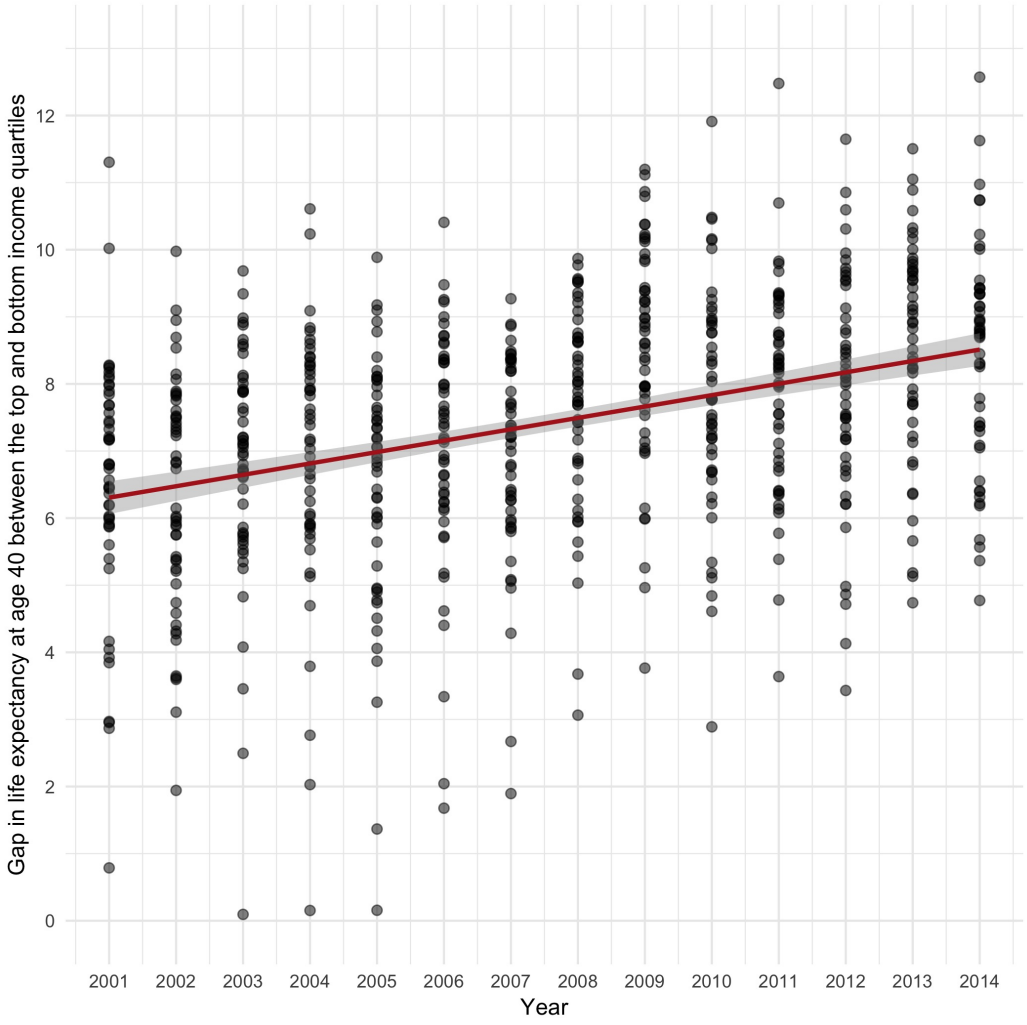

Figure 6.1: *Linear trend in the gap in life expectancy between the top and the bottom income quartiles between 2001 and 2014.*

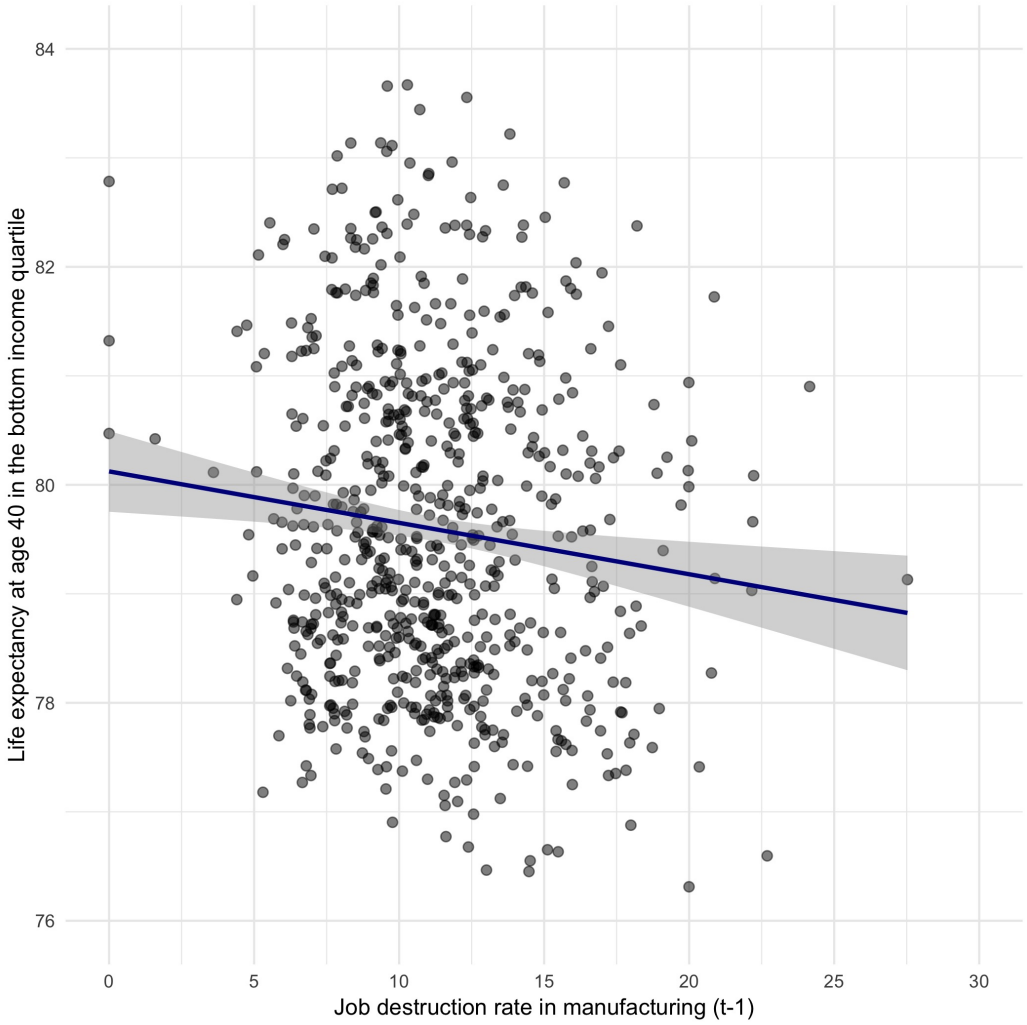

Figure 6.2: *Bivariate association between job destruction rate, lagged one year, and life expectancy at age 40 in the bottom income quartile, 2001–2014.*

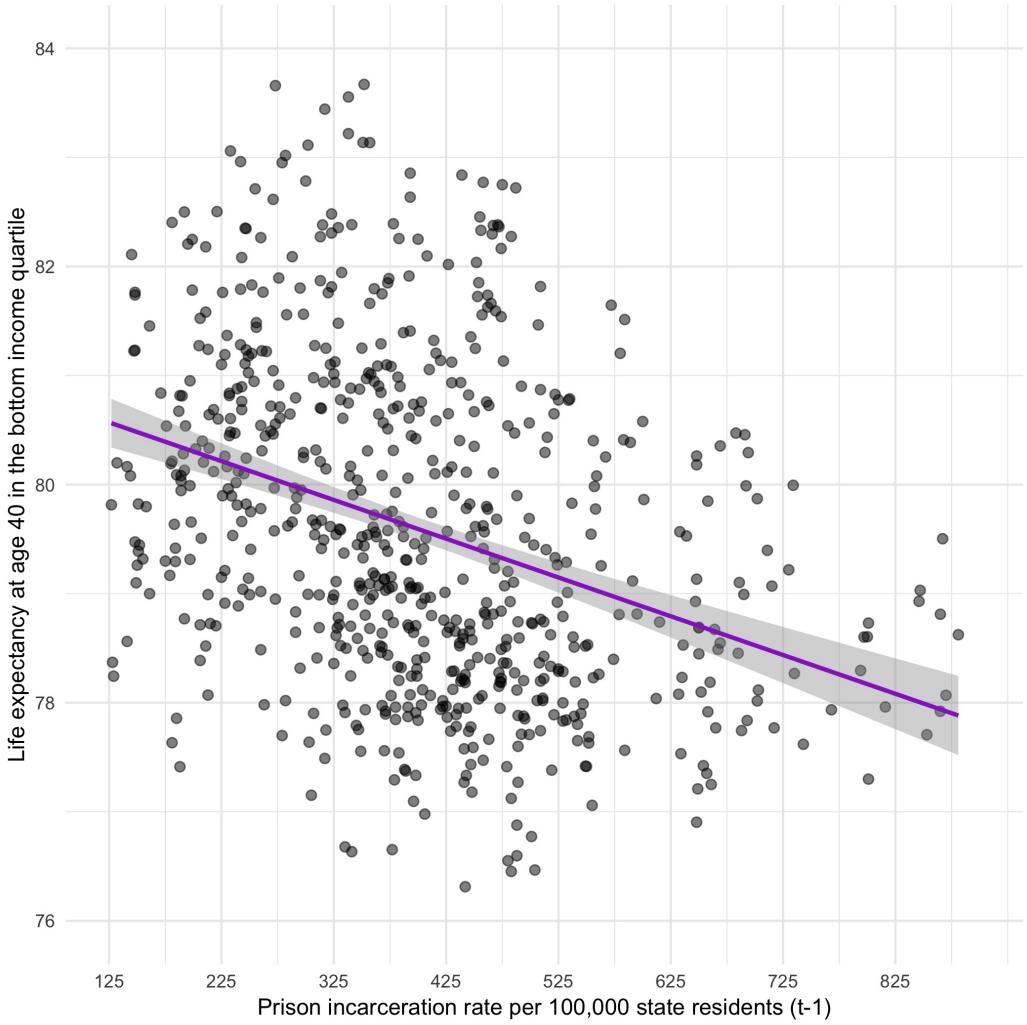

Figure 6.3: *Bivariate association between prison incarceration rate, lagged one year, and life expectancy at age 40 in the bottom income quartile, 2001–2014.*

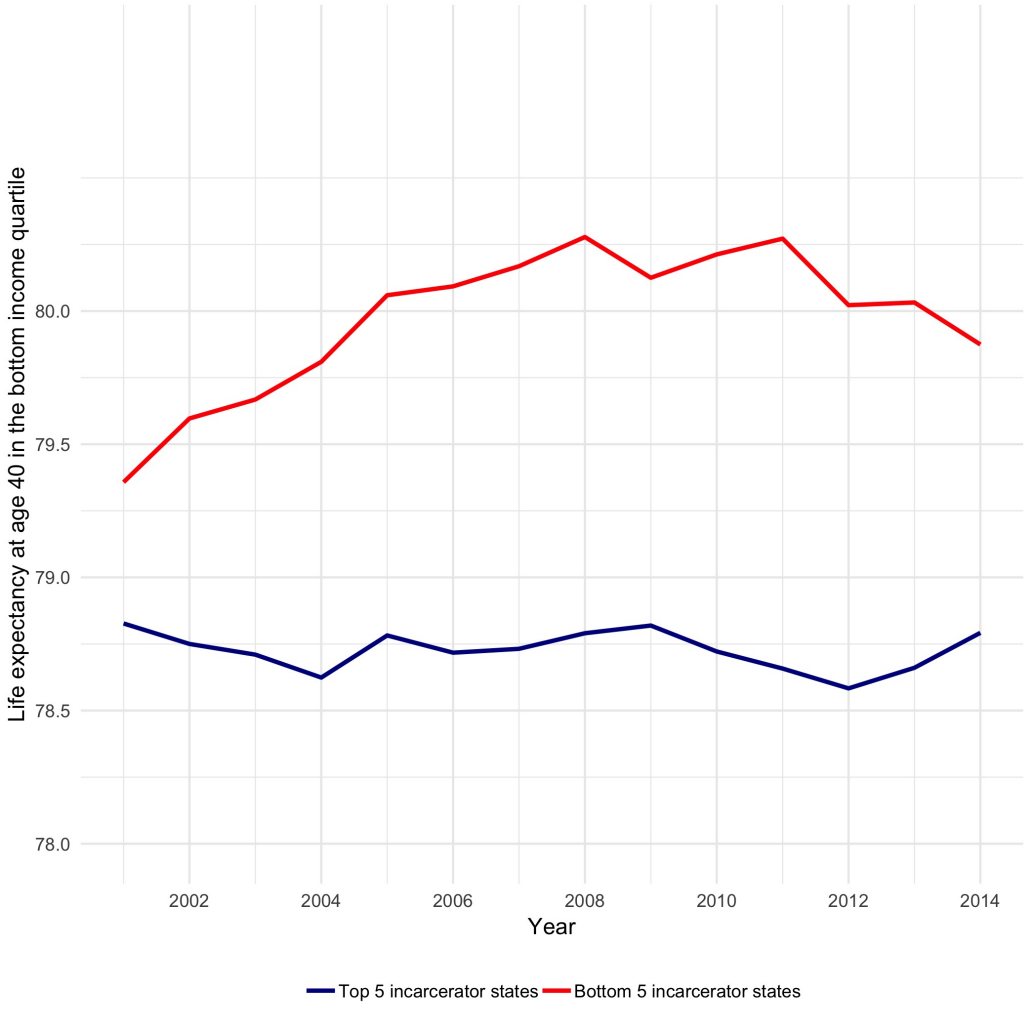

Figure 6.4: *Average life expectancy in the bottom income quartile in the top-five and bottom-five incarcerator states, 2001–2014.*

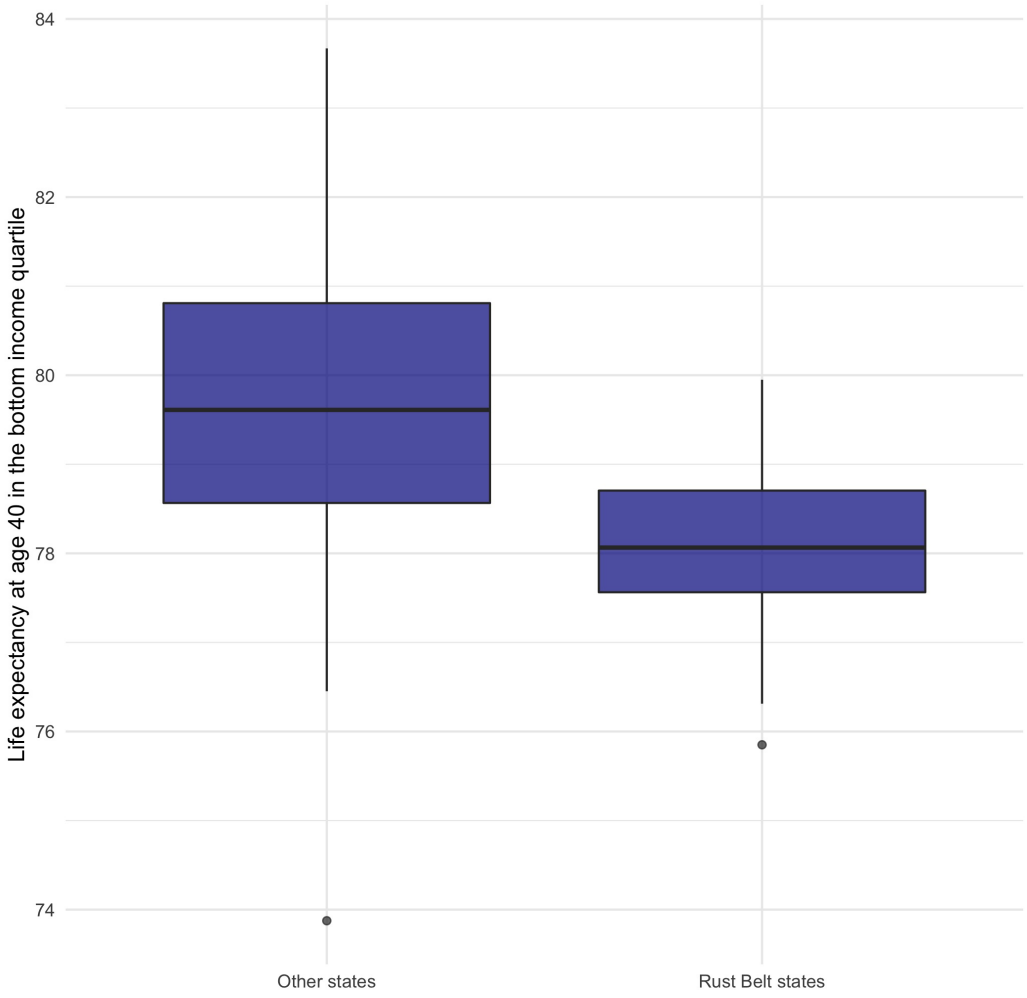

Figure 6.5: *Life expectancy in the bottom income quartile in Rust Belt states versus other states, 2001–2014.*

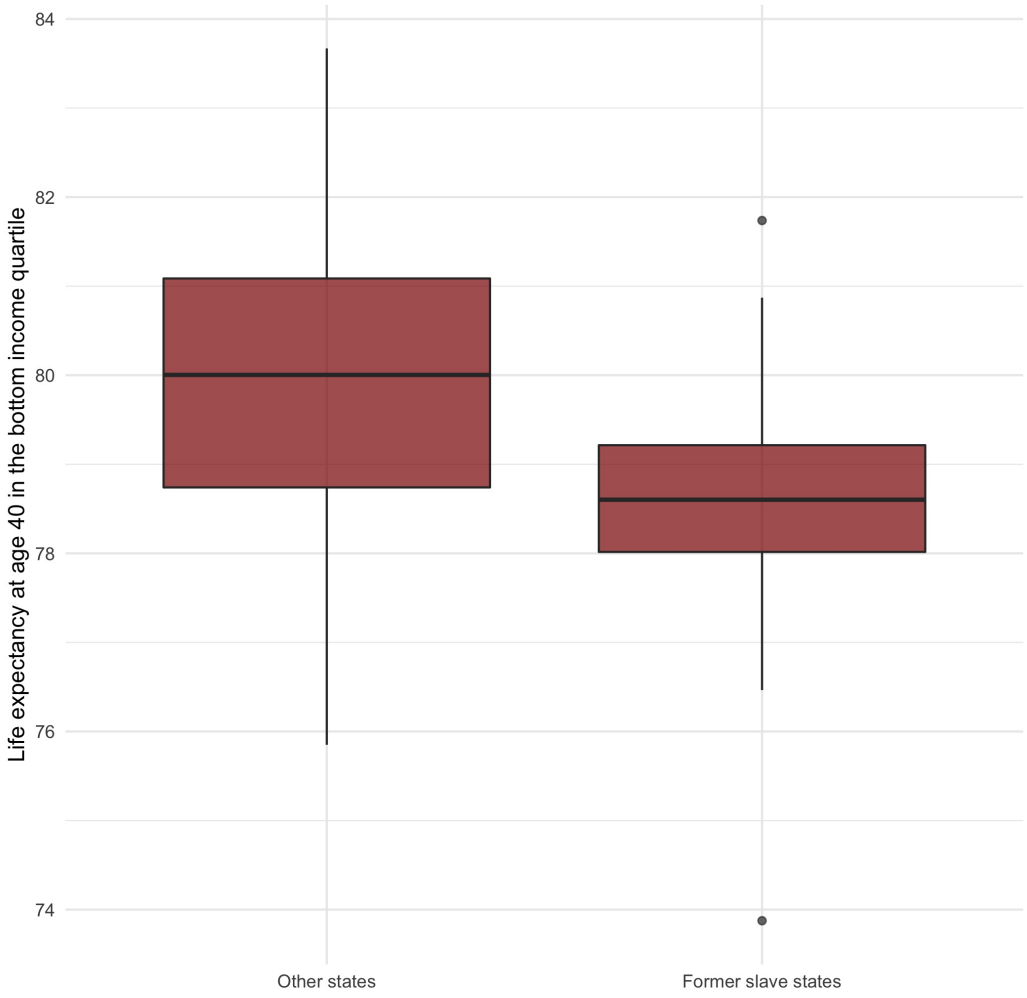

Figure 6.6: *Life expectancy in the bottom income quartile in former slave states versus other states, 2001–2014.*

Table 6.3: Baseline fixed effects model of life expectancy in the bottom income quartile

	Coefficient	Standard error	P-value
Job destruction rate $_{t-1}$	-0.26	0.08	0.002
Incarceration rate $_{t-1}$	-0.68	0.19	< 0.001

Notes: The outcome variable is state-level life expectancy at age 40 for those in the bottom income quartile; the predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year; the model only controls for aggregate annual time trends using year dummies (not displayed); both predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 697$. $R^2 = 24.4\%$.

residents) lost more than two years of life expectancy. The model meets all diagnostic criteria and explains almost a quarter of the state-level variation in life expectancy amongst the poor, as evidenced by an R^2 value equal to 24.4%.

The sensitivity analysis where state-level control variables are added and removed from the baseline model one by one is visualised in Figure 6.7. First of all, I run the same baseline model with race-adjusted life expectancy as the outcome variable. These estimates "remove the differences in life expectancy across areas and income groups that are due to differences in the racial composition of those areas" (Health Inequality Project, 2016). The results prove

robust to such differences, and also to all other control variables, as evidenced by how the magnitudes and confidence intervals of deindustrialisation and incarceration remain largely unchanged across alternative model specifications. The majority of the controls are statistically insignificant, but a truly remarkable result is that living in rich states or states undergoing economic growth does not aid the poor, and may even have a negative effect (β_{GDP} = -0.37; 95% CI: [-0.65, -0.10]; $P = 0.008$).[1] Otherwise, a high population fraction of smokers is, as expected, associated with a reduced life expectancy (β = -0.19; 95% CI: [-0.34, -0.03]; $P = 0.02$).

As a robustness check, I also bundle different control variables together into three main categories: behavioural controls, economic controls, and welfare state controls. Results for these models are displayed in Tables 6.4, 6.5, and 6.6. This does not change the substantive findings. Once again, job destruction and incarceration rates are statistically significant across all models, whereas most control variables remain insignificant.

When I run similar models with life expectancy in the *top* income quartile as the outcome variable, the impacts of deindustrialisation and incarceration are negligible, as shown in Table 6.7 and Figure 6.8. However, the reader will note that both GDP per capita and economic growth exert a significant positive impact ($\beta_{GDP} = 0.50$; 95% CI: [0.09, 0.92]; $P = 0.02$; $\beta_{growth} = 0.26$; 95% CI: [0.12, 0.40]; $P < 0.001$). When viewed in tandem with the results for the bottom income quartile, this reflects the inegalitarian nature of American

[1] Perhaps this can be seen as yet another sound of the death knell for McKeown's thesis, according to which economic growth is *the* fundamental force for improving health and wellbeing (see Szreter, 2005).

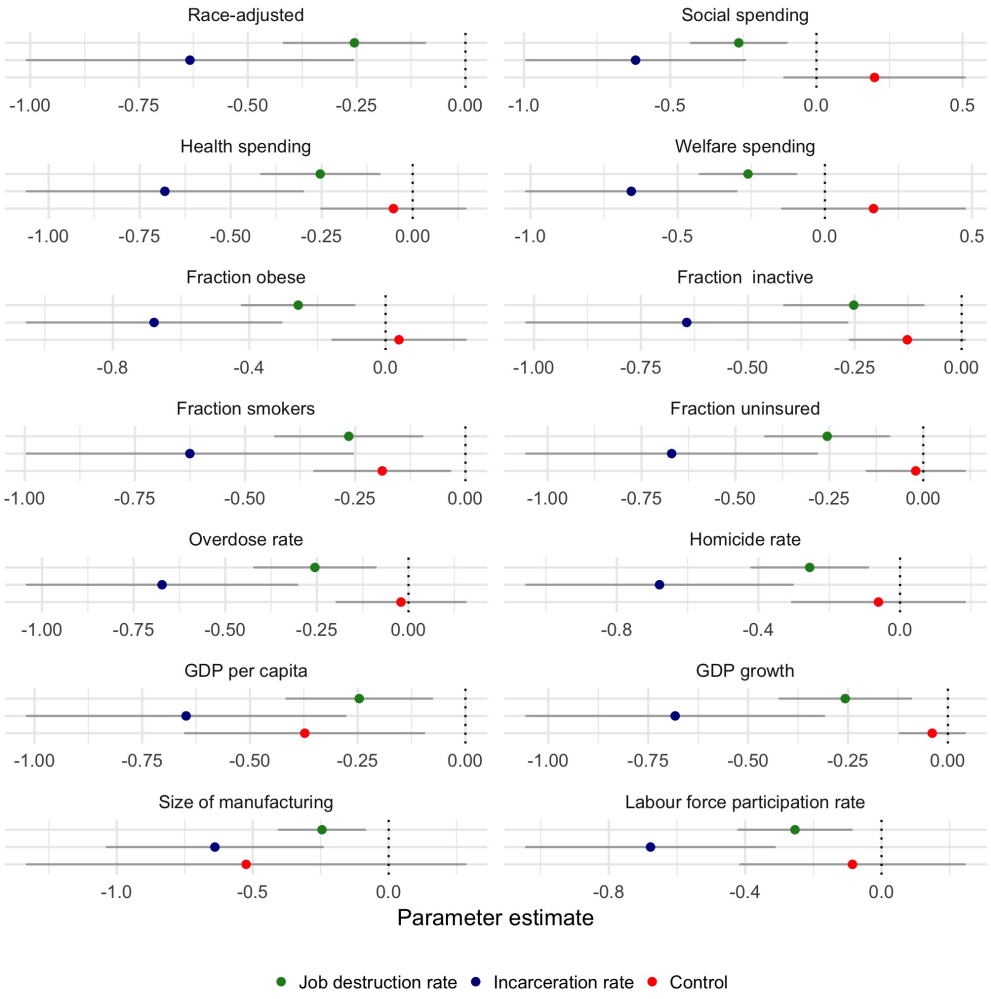

Figure 6.7: *Coefficient plot of fixed effects control models; the outcome variable is state-level life expectancy at age 40 for those in the bottom income quartile; the main predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year, adjusted for aggregate annual time trends using year dummies (not displayed); control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor.*

Table 6.4: Fixed effects model with behavioural controls

	Coefficient	Standard error	P-value
Job destruction rate $_{t-1}$	-0.26	0.08	0.002
Incarceration rate $_{t-1}$	-0.60	0.20	0.002
Fraction obese	0.03	0.10	0.77
Fraction physically inactive	-0.12	0.07	0.10
Fraction smokers	-0.17	0.08	0.04
Overdose rate	-0.01	0.08	0.94
Homicide rate	-0.03	0.12	0.77

Notes: The outcome variable is state-level life expectancy at age 40 for those in the bottom income quartile; the predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year; the model controls for the state population fraction who are obese, physically inactive, or smokers, the state overdose and homicide rates, as well as for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 691$. $R^2 = 25.0\%$.

Table 6.5: Fixed effects model with economic controls

	Coefficient	Standard error	P-value
Job destruction rate $_{t-1}$	-0.23	0.08	0.005
Incarceration rate $_{t-1}$	-0.60	0.20	0.003
GDP per capita	-0.41	0.22	0.06
GDP growth	-0.004	0.03	0.90
Size of manufacturing	-0.37	0.41	0.37
Labour force participation rate	0.05	0.19	0.81

Notes: The outcome variable is state-level life expectancy at age 40 for those in the bottom income quartile; the predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year; the model controls for state GDP per capita, GDP growth, the size of the manufacturing sector, labour force participation rates, as well as for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 647$. $R^2 = 26.4\%$.

Table 6.6: Fixed effects model with welfare state controls

	Coefficient	Standard error	P-value
Job destruction rate $_{t-1}$	-0.26	0.08	0.001
Incarceration rate $_{t-1}$	-0.59	0.20	0.003
Social spending	0.16	0.16	0.32
Health spending	-0.06	0.10	0.56
Welfare spending	0.13	0.17	0.43
Fraction uninsured	0.00004	0.06	1.00

Notes: The outcome variable is state-level life expectancy at age 40 for those in the bottom income quartile; the predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year; the model controls for state social spending, health spending, and public welfare spending, the state population fraction with no medical insurance, as well as for aggregate annual time trends using year dummies (not displayed); all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 691$. $R^2 = 24.8\%$.

Table 6.7: Baseline fixed effects model of life expectancy in the top income quartile

	Coefficient	Standard error	P-value
Job destruction rate $_{t-1}$	0.07	0.16	0.65
Incarceration rate $_{t-1}$	-0.27	0.26	0.30

Notes: The outcome variable is state-level life expectancy at age 40 for those in the top income quartile; the predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year; the model only controls for aggregate annual time trends using year dummies (not displayed); both predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor. $N = 697$. $R^2 = 41.7\%$.

growth, which seems to benefit the wealthy but which does little, if anything, to relieve the plight of the worst off.

6.4 Discussion

The main findings suggest that, between 2001 and 2014, the loss in life expectancy for the bottom income quartile associated with deindustrialisation and incarceration was substantial. To put the results in perspective, the demographic impact of all cancers corresponds to approximately 3.2 years of reduced life expectancy (Arias et al., 2013). On the basis of the findings in this chapter, the implied average gain, were incarceration and deindustriali-

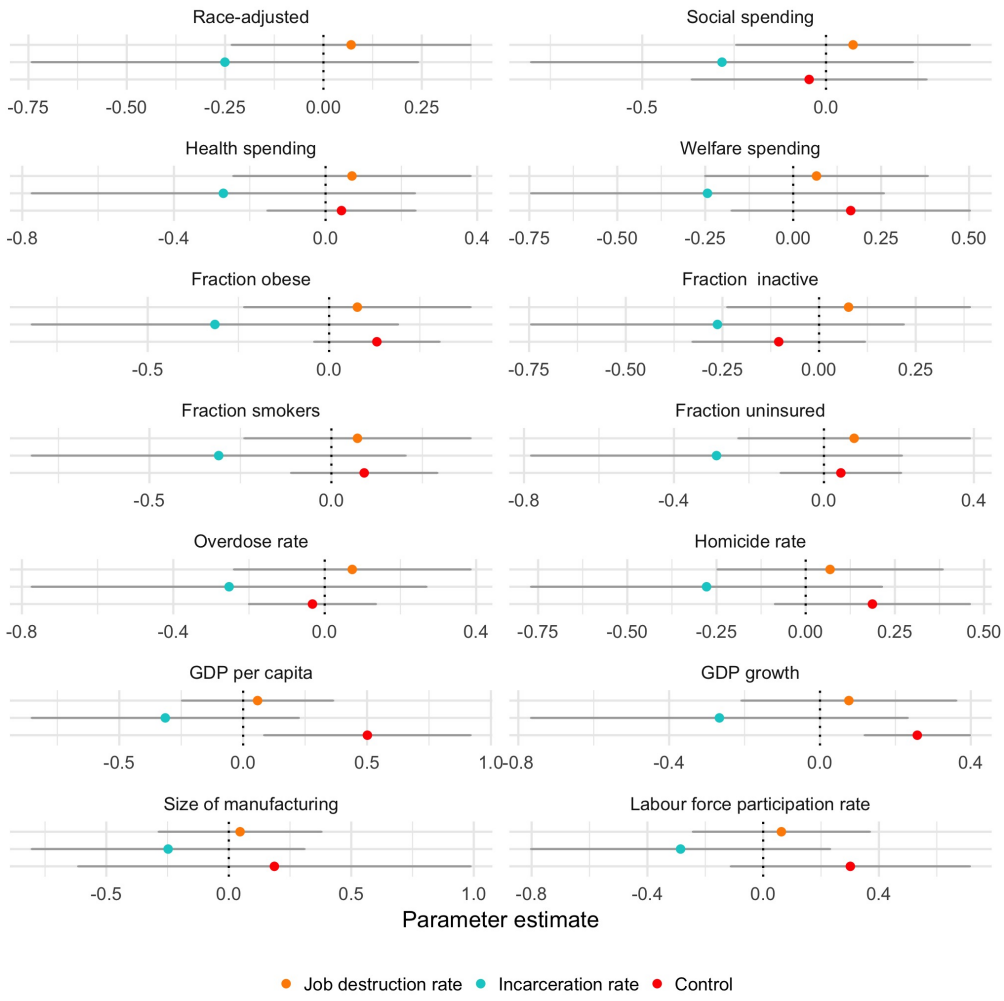

Figure 6.8: *Coefficient plot of fixed effects control models; the outcome variable is state-level life expectancy at age 40 for those in the top income quartile; the main predictors are the annual job destruction rate in the manufacturing sector and the prison incarceration rate per 100,000 population, both variables lagged one year, adjusted for aggregate annual time trends using year dummies (not displayed); control variables are added and removed one by one; the figure shows parameter estimates and corresponding 95% confidence intervals; all predictors are standardised by calculating deviations from the variable mean and dividing by one standard deviation; coefficients are interpreted as the change (in years) in life expectancy associated with a one standard deviation increase in each predictor.*

sation to be entirely eliminated (although an extreme counterfactual), would be over two and half years. This suggests that the adverse health effects of rapid socioeconomic dislocation and of the punitive regulation of poverty could explain virtually the entire increase in the vital gap between the top and the bottom income quartiles since 2001, which has increased by around two years (see Figure 6.1).

As noted earlier, it is likely that these phenomena unleash cascading effects: the weakening of American labour has left large swathes of the population in chronic unemployment, vulnerable to economic insecurity, psychosocial stress, and behavioural patterns involving drug abuse, self-harm, or interpersonal violence (Matthews et al., 2001; Kubrin and Weitzer, 2003; Kubrin et al., 2006; Browning and Heinesen, 2012; McLean, 2016). Moreover, it is plausible to suggest that smoking, physical inactivity, overweight/obesity, and other proximal determinants may be viewed as pathways rather than confounders of the relationship between deindustrialisation and life expectancy. However, the health behaviour variables used in the present analysis are aggregated up to the state level from the Behavioral Risk Factor Surveillance System surveys and do not provide the empirical basis to explicitly test this hypothesis.

The political response to this form of social turbulence has been largely punitive, as was detailed in the Introduction and chapter 2, further perpetuating and amplifying inequalities in life expectancy. A further consideration is that, in areas with lower life expectancy, individuals may reason that there is little point in investing in measures that would improve their economic prospects and may substitute short-term rewards, even if illegal, for uncer-

tain longer-term benefits, consistent with a substantial body of evidence on time preferences and health-related behaviour (Griskevicius et al., 2011; Barlow et al., 2016). Thus, deindustrialisation, incarceration, and poor health mutually interact to create a vicious downward cycle.

I acknowledge the limitations of this analysis. The spatiotemporal dimensionality of the data imposes restrictions on the statistical power of the presented models. Significant portions of variance are suppressed in a state-level analysis, which conceals deeper inequalities and more salient effects located at the county or city levels, as revealed in the previous chapters. The time period in question (2001 to 2014) comes well after the onset of massive industrial decline and the explosion of incarceration that started in the 1970s – although, as noted in chapter 2, there was a sharp acceleration in employment decline in manufacturing beginning in 2000. As such, my analysis fails to capture the full magnitude of the effects of interest.

The data from the Health Inequality Project report lower mortality rates than those registered by the Social Security Administration. For methodological reasons, Chetty and colleagues restrict their sample to individual residents with positive earnings (any income subject to filed tax returns). As they point out in their web appendix, the 9% of the population who are thus excluded from their analysis account for no less than 38% of total deaths. This means that the average mortality rate in this fraction of the population is at least four times larger than the mean mortality rate of individuals with positive earnings. As such, the present analysis does not capture the impacts of deindustrialisation and incarceration on those who fall below the positive income threshold. One may surmise that both factors, but incarceration in

particular, exert a substantial deleterious effect on the life chances of these individuals. Another limitation is that life expectancy data by income have only been released at age 40, thereby excluding deaths at younger ages, for example from drugs and violence, that may be especially important in this population, as was indicated in the earlier analysis of premature mortality risk. Indeed, spatial variation in overall life expectancy at birth – as was used in the previous chapter – is far greater than (state-level) variation in income-disaggregated life expectancy at age 40. In short, the health consequences of industrial decline and punitive social policy are likely underestimated with currently available data.

6.5 Conclusion

Between 2001 and 2014, deindustrialisation and incarceration constituted important determinants of life expectancy for the poor but not for the wealthy, generating deeply consequential health deficits for states adopting punitive responses to economic stagnation. The historical legacies of rapid industrial decline and slavery are likely to exert substantial long-term effects on vital inequality. Therefore, for a full understanding of health inequalities in the United States, researchers must remain conscious of the upstream political and economic determinants of public health. If public policy responses to growing health inequalities are to be effective, they must consider industrial policy as well as ending (hyper)incarceration of society's most vulnerable.

Conclusion

> *The libidinal achievements demanded of an individual behaving as healthy in body and mind, are such as can be performed only at the cost of the profoundest mutilation [...]. Just as the old injustice is not changed by the lavish display of light, air and hygiene, but is in fact concealed by the gleaming transparency of rationalized big business, the inner health of our time has been secured by blocking flight into illness without in the slightest altering its aetiology. [...] The very people who burst with proofs of exuberant vitality could easily be taken for prepared corpses, from whom the news of their not-quite-successful decease has been withheld for reasons of population policy. Underlying the prevalent health is death.*
>
> Theodor Adorno, *Minima Moralia*

"All that is solid melts into air", Marx and Engels famously said. Capitalism's "creative destruction" once left over half a million handloom weavers to die a lingering death "when industry no longer had any need for them"

(Hobsbawm, 1996: 37). Today, the industrial proletariat, two centuries after it was "introduced to its role not so much by attraction [...] but by compulsion, force and fear" (Pollard, 1965: 207), is crumbling under the weight of economic restructuring. Much like its birth, it seems to be dying in agony, and in so doing, to quote Marx once more, is experiencing "the loss of [the] old world with no gain of a new one" (cited in Hobsbawm, 1995: 162). It is therefore important to preempt any "creeping industrial nostalgia" (Cowie and Heathcott, 2003: 14) when discussing the political economy of America's public health landscape (see Milkman, 1997). Indeed, industrialisation and deindustrialisation are "merely two ongoing aspects of the history of capitalism that describe continual and complicated patterns of investment and disinvestment" (Cowie and Heathcott, 2003: 15). The story of (de)industrisalisation is thus not "a simple, unidirectional story of political and economic stability followed by decline" but one that is "pockmarked with explosions, relocations, desertions, and competitive struggles" (*ibid*: 14). The same goes for the disciplinary treatment of poverty, which, despite the incomparable expansion of the penal apparatus in recent decades, has a rich historical foundation (Piven and Cloward, 1993; Geremek, 1994). This is why, throughout the thesis, I have tended to employ the word "capitalism" rather than the more in-vogue notion of "neoliberalism". Although the temporal window of my analysis has largely been confined to the 1980s onwards, an implicit argument of mine is quite simply that the historical roots of the contemporary patterning of life chances go back much further than a sole focus on neoliberalism would indicate. Although the distinct institutional features of neoliberalism have been studied extensively and their

deleterious effects on health have been documented (e.g. Labonté and Stuckler, 2016; see Beckfield and Krieger, 2009), its frequent usage easily morphs into a discursive bulwark against a critical (re)conceptualisation of capitalism itself.[2]

My substantive argument has been that the currently prevalent mode of poverty regulation in the wake of economic decline is characterised by how political emphasis is consistently shifted from the protective to the corrective wing of the policy apparatus. Social assistance for the victims of "creative destruction" is subject to increasingly stringent conditions, chief amongst them the forced participation in precarious wage work in segmented labour markets (see Kalleberg, 2011). At the bottom of the class structure, American capitalism thus seems to have reinstituted in naked form what Weber identified as one of its integral components, namely "free labour" whereby workers, "under the compulsion of the whip of hunger" (Weber, 2013: 277), sell their labour power. The gaping wounds inflicted by economic disruption are sutured with disciplinary policy instruments that either enforce degrading work parameters or invisibilise those who are caught in the interstices through penal confinement and sociopolitical ostracisation. Taken together, deindustrialisation and incarceration have fractured the working class and contributed to the rise of a precarious (sub)proletariat in which health disadvantages are disproportionately concentrated. The result is a nascent "pre-

[2] Of the word "capitalism", Fernand Braudel notes that "after a long struggle, I gave up trying to get rid of this troublesome intruder. I decided in the end that there was nothing to be gained by throwing out along with the word the controversies it arouses. [...] In any case, such precautions are delusive: if capitalism is thrown out of the door, it comes in through the window" (Braudel, 1983: 231).

cariat" suffering from joint social and biological "sub-citizenship" (Sparke, 2017).

I have sought to substantiate this line of argument in two principal steps. First, I have sketched the historical backdrop against which American social policy has undergone a distinctively punitive transformation. On the one hand, I have argued that the political economy of industrial decline, and the concentration of corporate power that subtends it, fed into deepening vulnerability at the bottom of the class structure. Most notably, the nexus of class and ethno-racial division heralded a prolonged period of intense political turbulence in the wake of rapid economic change. On the other hand, this very development opened the space for a punitive rather than protective policy response from above, driven by an ideological mechanism by which the fundamentally *political* issue of deepening inequality was recast as a problem of "law and order" – i.e. one that required the iron fist of the penal state rather than the maternal hand of the welfare apparatus. Buttressed by media sensationalisation and consolidated by a large-scale electoral realignment amongst Democrats and Republicans alike, this punitive turn discarded any mainstream political commitment to public investments in housing, (mental) health care, employment security, or the "root causes" of crime and violence. Instead, it reoriented welfare towards workfare and initiated a historically unprecedented expansion of the criminal justice system.

Second, I have presented a series of empirical analyses drawing on hitherto unavailable data at the county level between 1980 and 2014. Using a number of methodological approaches, I have demonstrated an empirically robust association between high rates of incarceration and disparities in health

and wellbeing. I commenced with the analysis of mortality rates from drug use disorders, which have grown exponentially in recent times, taking tens of thousands of lives every year. Both fixed effects, random effects, and "between"-county models showed that jail and prison admissions rates may constitute important predictors of America's ongoing public health crisis. Similarly robust associations were demonstrated with broader measures of vital inequality, namely age-standardised all-cause mortality rates, premature mortality rates, and life expectancy at birth.

The limitations of the present study have been listed earlier but are worth revisiting as a means of delineating avenues for future research. First, my principal focus has been on incarceration, but, as emphasised throughout, incarceration is but one component of a much broader policy repertoire. There is a need to unpack the socially differentiated operations of the policy apparatus, which, as noted in chapter 2, mobilises different "hands" of the state in divergent ways across different social strata, from condemning deprived households to homelessness through eviction to ensuring generous welfare provisions for the wealthy through large-scale public expenditures. Future research in the political economy of public health should seek to probe the causes and consequences of that distinct form of institutional imbrication that results in retributive interventionism at the bottom and avuncular protectionism at the top of the social structure.

Second, although I have posited the existence of a causal chain leading from the macroeconomic to the molecular, I have not been able to empirically disaggregate the relevant pathways. This is primarily a data problem, but also involves theorising the relevant "fundamental" causal relationships (Link

and Phelan, 1995). A more complete analysis would involve fleshing out the mediators leading from political and economic factors such as industrial decline to population health outcomes, but also to investigate treatment effect heterogeneity across social and physical space. Chapter 6 showed that industrial decline and incarceration affect the lifespans of the poor but not the rich. However, there are other forms of heterogeneity that warrant deeper examination, including interactive spillover effects across a range of individual-level and local attributes, notably at the interface of the three main types of inequality. For instance, how does hyperincarceration in one community affect a neighbouring community? (How) does "despair" spread through social networks? How does resource deprivation "spill over" onto symbolically mediated gradations of honour and status (say, ethno-racial domination), and *vice versa*? Or how does the gendered nature of punitive social policy, whereby men go to prison and women are thrust into precarious wage work or evicted, relate to the fact that women systematically outlive men, especially at the lower end of the socioeconomic order? Answering such questions entails not only access to better data but also a deeper engagement with the theory of causality – one which identifies (in the statistical sense) operant mechanisms but also accommodates feedback loops and recursive relations (such as the ones between racialised poverty, incarceration, and health).

Third, the principal unit of analysis in this thesis, the county, has not been problematised in a systematic way. The main justification for using the county measure has been that, contrary to common belief, it constitutes the principal unit of the American penal state. Even those who are sentenced to long prison sentences at the state level are, in the vast majority of cases,

passed through local jails in pre-trial detention. However, there is a significant amount of variation in how county authorities operate their jails and how the local penal apparatus relates both to state-level institutions and to local populations. An effort has been made in the preceding analyses to prune the data set in ways that facilitate easy comparability across counties, yet future research should further probe the kinds of regional differences at work and relate them to the lived experiences of local populations. This warrants the use of disaggregated data that can assess the criss-crossing of individual-level and ecological forces within a truly multilevel framework.

Fourth, in the vein of Case and Deaton, the thesis has placed particular emphasis on the "demand-side" factors that have driven the overdose epidemic – factors like (political responses to) socioeconomic insecurity. However, a major focus in the extant literature is on "supply-side" factors, most notably the role of pharmaceutical corporations like Purdue, who manufacture and aggressively advertise opioids like OxyContin through a variety of channels, including via physicians. In chapter 4, I sought to account for this aspect by matching counties on local opioid prescription rates. However, this is unlikely to do justice to the causal complexity at work, nor does it take into account other dimensions of local drug environments, such as the availability of illicit drugs like cocaine or heroin, which play an important substitute role once addiction has been created. Broader vital inequality (in all-cause mortality or life expectancy) is also shaped by the availability of (un)healthy food and drink, tobacco and alcohol, or physical exercise facilities – all of which have their own corporate determinants. In short, future work on this topic must better investigate the interplay between "supply" and "demand"

factors.

Fifth, the present study has failed to assess the temporal structure by which macro-level factors shape vital inequality. In epidemiological parlance, the "aetiological period" has not been adequately addressed. Moreover, there may be causal heterogeneity across time periods. My models adjust for aggregate time trends using a maximally flexible year term, namely year dummies, which helps reduce model dependence related to temporal heterogeneity but suppresses temporally mediated differences that may be of substantive interest, including for instance changes in demographic compositions and (im)migration patterns. However, there is heterogeneity not only across time but also across space in ways that do not necessarily match onto one another, rendering the choice of cut-off points and county-year groupings somewhat arbitrary. I have therefore refrained from further time-stratification, which is at least partly predicated on a precise knowledge of the aetiological windows at work, including long-run effects. Future research should seek to integrate an understanding of timing that goes beyond the addition of "lags" in statistical models (Beckfield, 2018) or simply "adjusting" away a quantity of substantive interest, namely time and its autocorrelating effects on observed data (see King, 1998: 163). As with each of the aforementioned points, such a task is obviously predicated on the existence and availability of high-quality and relevant empirical materials, ideally in the form of micro-level administrative records that capture an entire population over time (see e.g. Chetty et al., 2018), coupled with the integration of biomedical data.

Sixth, I have made the case that vital inequality in the United States is anchored in a set of social dynamics that operate across purely ethno-racial

boundaries. Nevertheless, a limitation of my analysis has been a lack of deeper engagement with the nexus of class and race at the empirical level. My models control of the ethno-racial composition of counties, as well as for the median household income, but ultimately fail to move beyond the deployment of seemingly isolated (and imperfectly measured) "variables" in analysing the political economy of inequality. This limitation is not reducible to that of "quantitative" methods per se, as evidenced by how the use of administrative records has proven fruitful in quantifying some of the relevant dynamics at work (see e.g. Chetty et al, 2018). Nonetheless, an ethnographic approach may very well have shed fresh light on this matter in a way that eludes even the most refined statistical model.

Seventh, where the present study has been entirely focused on the United States, a comparative political economy of public health may breathe fresh understanding into this nascent field of research. A break with "methodological nationalism" would involve the study of how different configurations of power exert diverse health impacts across "varieties of capitalism" (Hall and Soskice, 2001) over time and space. However, as evidenced by the history of deindustrialisation, the political economy of capitalism is also the political economy of *global* capitalism, involving the world polity as a locus of political struggle between unequally equipped actors (Beckfield, 2003; 2010). As such, a promising avenue for future research is the study of how a distinct policy paradigm has not only spread across the Atlantic to Europe (see Nosrati and Marmot, 2019) but has also been imposed on low- and middle-income countries at the behest of powerful international financial organisations like the International Monetary Fund or the World Bank (Kentikelenis, 2017).

The imposition of structural adjustment – typically entailing a set of mandated economic reforms targeting fiscal austerity and market "deregulation"[3] – typifies one form of coercive norm enforcement of which the ultimate manifestation is armed intervention. The latter is a remarkably underdeveloped area of research, i.e. the study of military power as a determinant of public health (cf. Murray et al., 2002). In this context, the United States constitutes a valid starting point of enquiry – being, as it is, a country whose unparalleled military zest reached its latest climax under the presidential aegis of a Nobel Peace Prize laureate.

Finally, there is a need to complement the study of "unequalisation" with that of "equalisation", as seen through a macroscopic lens. This involves investigating structural forces and identifying concrete mechanisms by which (in)equality is reworked or recalibrated. Although the principal tools of equalisation in an unequal world may be known (Therborn, 2013: 64, Table 5), their operationalisation, at least on a global scale, is inchoate at best. Equalisation implies not only the development of roadmaps to policy interventions, which poses challenges in its own right (Reeves, 2017). It also implies a fundamental reworking of the very parameters of our collective imagination. At a time when, against the backdrop of imminent climate breakdown, the most widely espoused solution to all the world's problems is continued economic growth, it is becoming increasingly hard to envision a qualitatively different yet politically potent *modus operandi*. This is a symptom of how, as it were, "it is easier to imagine the end of the world than

[3] A more accurate term is "*re*-regulation" (in favour of corporations).

the end of capitalism".[4] Moreover, insofar as equalisation is the obverse of unequalisation – an effort to heal an already inflicted wound or to console the already traumatised – a commitment to equality is perhaps even better served by addressing the underlying aetiology of our collective predicament. Perhaps, then, the first step towards veritable equality is a reinvented fidelity to the principle of justice.

[4] Attributed to both Fredric Jameson and Slavoj Žižek. See Fisher (2009).

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